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THE INFERIOR ESOPHAGEAL CONSTRICTOR IN RELATION TO LOWER ESOPHAGEAL DISEASE

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IT IS interesting to note the lack of definite information and the conflicting opinions relative to the anatomy and physiology of the esophagus and to the pathogenesis of esophageal diseases such as the Plummer-Vinson syndrome, esophagospasm, achalasia or mega-esophagus, pulsion diverticula, and idiopathic strictures. This lack of knowledge results from the inability either to produce such conditions experimentally or at least to correlate experimental and clinical findings. It is also difficult or impossible to deduce the genesis of pathologic conditions from gross and microscopic findings of well-established chronic lesions. It is also difficult or impossible to determine whether such findings may be primary and part of the etiology or entirely secondary manifestations. In view of this situation observations relative to these conditions should be presented by those interested in this field in an attempt to help clarify our understanding and perhaps help as an eventual aid in therapy.

The esophagus has a well-developed outer longitudinal muscle layer and a less well-developed inner muscle layer. The longitudinal muscle extends from the cricoid cartilage and cricopharyngeus muscle above to insert below in an interlacing manner into the middle and outer muscle layers of the cardia. The inner muscle layer commences in the upper end independently of other groups of muscle fibers. The arrangement of these fibers varies in the different segments of the esophagus. They run in an elliptical direction around both sides of the esophagus and downward in the upper third; they are circular for a small segment in the middle third, below which they again run in an elliptical course in a downward direction toward the posterior aspect.

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In the lower third the muscle fibers wind about the esophagus in a spiral manner. The inner muscle layer continues in the inner and middle muscle layers of the cardia and stomach. Laimer[1] and Lerche[2] described a circumscribed thickening of the inner muscle layer approximately 1 to 3 cm. above the diaphragmatic hiatus which they referred to as the lower physiologic constrictor or the inferior esophageal sphincter. Deeper longitudinal fibers from this sphincter insert into the longitudinal layer and have a dilating action on the sphincter. The roentgenologist is more familiar than the surgeon with the sphincterlike action present in the lower esophagus as noted during the barium swallow fluroscopically. Careful dissection of the inner muscle layer demonstrates areas where the arrangement of the spiral and elliptical fibers are such that the thickness of the inner muscle layer is considerably less than elsewhere and which, as will be shown later, may be weak points which are a factor in the development of pulsion diverticula. The location of such weak points may be varied which accounts for the different locations where pulsion diverticula may occur and the fact that they may be multiple. The presence of fibers in diverticula from the outer longitudinal muscle but not from the inner layer further suggests herniation through weak points in the inner muscle.

The muscular coordination of the esophagus in the act of deglutition is under the control of the parasympathetic and sympathetic nervous systems. When there is a preponderance of sympathetic control such as might occur when there is degeneration, reduction, or absence of ganglion cells of Auerbach's plexus or the vagus nerves, muscular coordination is impossible and the circular fibers of the inferior esophageal constrictor not only become contracted and hypertrophied to the degree of causing esophageal obstruction but also fail to relax and dilate before the oncoming esophageal peristaltic wave so that functional obstruction results.

Tamiya and Tamura[3] have shown that a preponderance of sympathetic control occurs when the ganglion cells of Auerbach's plexus are destroyed experimentally. Knight and Adamson[4] have also shown that vagal resection in the cat can produce functional stenosis of the lower esophagus with dilatation above similar to what is seen in achalasia or mega-esophagus (whichever term one prefers) which can be prevented by simultaneous section of the sympathetic fibers, or when produced can be relieved later by this procedure. Vagal denervation incident to either vagotomies or esophageal mobilization also causes contraction or spasm in the region of the inferior esophageal constrictor and dilatation above.

The presence of an inferior esophageal constrictor is best demonstrated in its hypertrophied state in patients with mega-esophagus (Fig. 1). Stimulation of the vagus nerves at the time of myotomy for mega-esophagus causes contraction of the esophageal musculature and relaxation of the sphincter in milder cases of mega-esophagus. No significant muscular response is seen following vagal stimulation in the far-advanced case, which suggests that the vagal dysfunction may be a secondary manifestation in the far-advanced case. This may be without effect because of possible loss of ganglion control as a

result of reduction or degeneration of Auerbach's plexus noted in many patients with mega-esophagus. The sphincterlike action in the terminal esophagus of patients with mega-esophagus can be demonstrated fluoroscopically to dilate under the influence of amyl nitrite and ergotamine. The dilating action of amyl nitrite on the sphincterlike mechanism of the hypertrophied circular muscle in the terminal esophagus has also been observed in some cases of achalasia at the time of surgical dissection.[5]

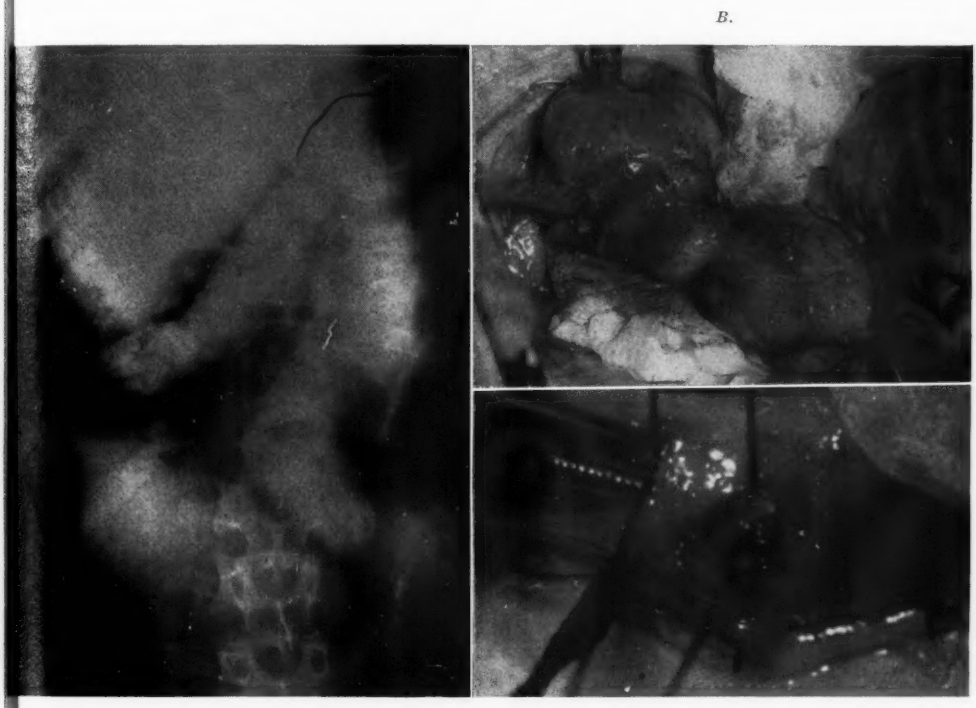


Fig. 1.—*A*, Esophagram of a patient with achalasia of the esophagus demonstrating the point of obstruction approximately 1 inch above the cardia at the site of the inferior esophageal constrictor. *B*, Surgical exposure of achalasia of esophagus demonstrating the site of inferior esophageal constrictor in its contracted state. *C*, Surgical dissection of the inferior esophageal constrictor.

Esophageal obstruction will cause secondary hypertrophy of both the longitudinal and inner muscle layer above the point of obstruction. The hypertrophy of the circular layer of the so-called inferior esophageal constrictor in patients with mega-esophagus far outproportions hypertrophy found elsewhere. The hypertrophied muscular constrictor or sphincter has been found in all fifteen patients with achalasia operated upon in the last two years during which time a search for this constrictor was made and a myotomy performed. Prior to this time a Heineke-Mikulicz or Finney cardioplasty was

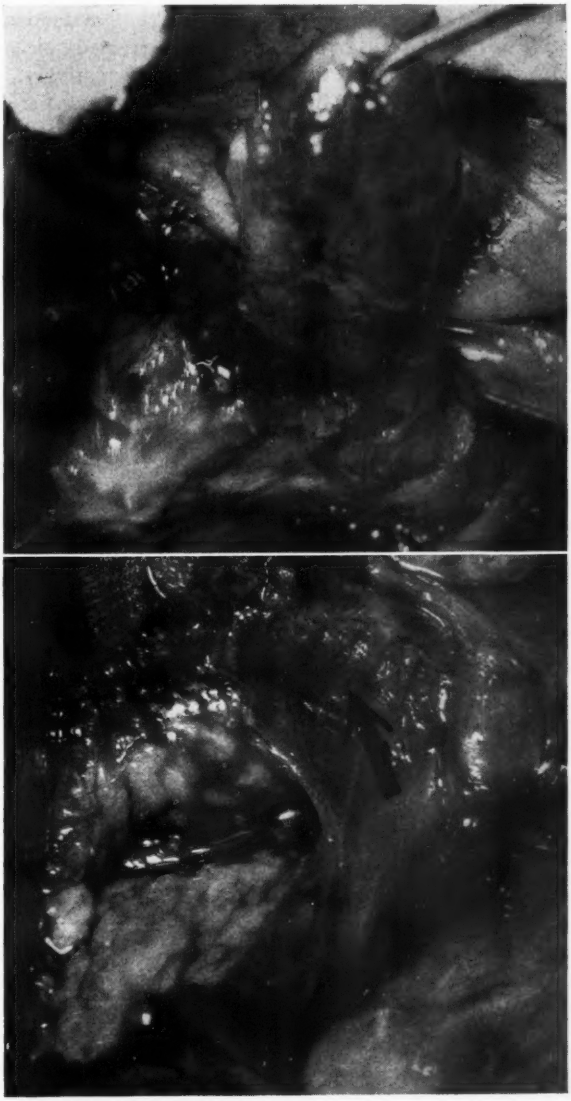
performed in thirty-two cases.[6] This procedure was discontinued because of the 28 per cent incidence of regurgitation esophagitis and bleeding. Three patients had partial esophagogastrectomies with satisfactory results, but it was found that an operation of this magnitude was not necessary. The fact that the symptoms of achalasia can be relieved by transecting the inferior esophageal constrictor further suggests that this muscular band is the predominant factor in the development of this condition.

Fig. 2.—Esophagram demonstrating pulsion diverticulum of thoracic esophagus and functional stenosis at the site of the inferior esophageal constrictor.

Functional dysphagia, esophagospasm, and the Plummer-Vinson syndrome may represent minor manifestations of this esophageal dysfunction and may be caused by a sympathetic imbalance and muscular incoordination associated with a hypertrophied inferior esophageal constrictor. Fortunately, symptoms are never severe enough in these conditions to warrant surgical exploration of this area. It also appears impossible to obtain detailed findings from postmortem material to confirm or disprove this theory. However, the similarity of functional dysphagia to mild to moderate degrees of so-called cardiospasm and the fact that both conditions are improved by esophageal dilatation leads one to conclude that the underlying mechanism may be the same.

The etiology of pulsion diverticula of the lower thoracic esophagus has never been satisfactorily given. Most authors have alleged that there is a congenital weakness of the esophageal musculature or that there is a weakness

A.



B.

Fig. 3.—*A*, Surgical exposure of a pulsion diverticulum. *B*, Arrow points to the transected hypertrophied circular muscle fibers of the inferior esophageal constrictor just distal to the stoma of the pulsion diverticulum.

due to a localized esophagitis. It is interesting to note that 65 per cent of the 126 patients with thoracic pulsion diverticula reported in the literature by Goodman and Parnes[7] had an associated cardiospasm. In the present series the majority had a temporary delay of barium in the terminal esophagus at the site of the inferior esophageal constrictor to a more pronounced degree than noted normally (Fig. 2) and three patients had a moderately severe cardiospasm with tortuosity (Fig. 6, *A*). I have treated ten patients with thoracic pulsion diverticula surgically and two conservatively. In seven a diverticulectomy was performed. In two there was an associated carcinoma necessitating an esophageal resection. One patient had both a diverticulectomy and partial esophagectomy performed because of a markedly tortuous esophagus. Symptoms of esophageal obstruction in the two remaining patients, one an 80-year-old man, have been relieved by periodic esophageal dilatation. Only six of these patients were operated upon during the past two years during which time search for a possible causative agent was made. The ages of the patients in this group ranged from 50 to 80 years of age. The older age incidence would discount any congenital theory of etiology unless one considers the weak areas in the inner muscle layer due to the anatomic arrangement of the muscle fibers as a congenital rather than an anatomic factor. Duration of symptoms ranged from two to eighteen years.

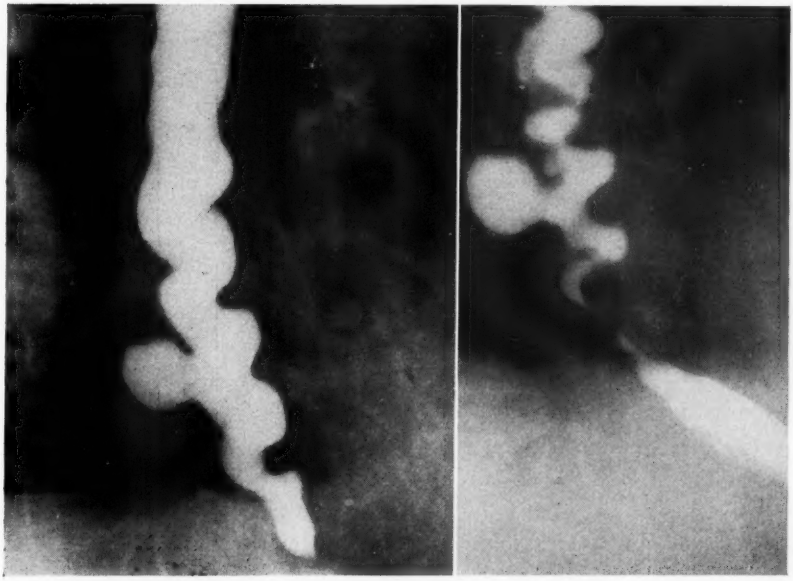
It was first noted several years ago that during the dissection of the neck of the diverticulum a hypertrophied band of circular muscle was present about the esophagus just distal to the mouth of the diverticulum. The obstructing nature of this hypertrophied band could be easily demonstrated by introducing a finger into the esophageal lumen. Further dissection of the band as demonstrated in Fig. 3 showed that it was similar to that noted in patients with achalasia. This hypertrophied circular band was transected and the esophageal opening closed with an inner mucosal layer of interrupted catgut and an outer muscular layer of interrupted 0000 Deknatel suture. Similar findings have been observed in the other patients.

Esophagoscopic examination in patients with pulsion diverticulum reveals esophagospasm or what might be referred to as functional stenosis in the terminal esophagus distal to the diverticulum. Fig. 4 demonstrates the pulsion diverticulum present in the 80-year-old patient who has been treated conservatively by esophageal dilatations. Tertiary contractions of the spiral muscle fibers of the inner muscle layer suggestive of esophageal obstruction are demonstrated radiographically. Possible other early diverticula are also developing as evidenced in the esophagrams. The fact that this patient's symptoms can be relieved by dilating the distal esophagus suggests that a hypertrophied inferior esophageal constrictor is the cause of the esophageal obstruction which in turn causes increased intraesophageal pressure which in turn causes the mucous membrane to herniate through weak areas in the inner muscle layer and pulsion diverticula to form.

Examination of the esophagus following esophagectomy for carcinoma in combination with the diverticulum (Fig. 5) showed that the circular muscle in the esophagus distal to the stoma of the diverticulum at the site of the inferior esophageal constrictor was more hypertrophied than the longitudinal

muscle; whereas, above the stenosis produced by the carcinoma there was generalized hypertrophy of both the circular and longitudinal layers to the same degree. The carcinomas in these cases were probably secondary to chronic irritation due to the diverticula. Microscopic examination of nerve fibers and the ganglion cells of Auerbach's plexus failed to show evidence of nerve degeneration in these two patients.

In the last case, a 77-year-old man with a history of dysphagia of twenty years duration, evidence of mega-esophagus and a pulsion diverticulum were both present. In addition to this there was a bilateral chronic pneumonitis due to many years of regurgitation and bronchial aspiration. Swallowing had



A. *B.*

Fig. 4, *A* and *B*.—Esophagrams demonstrating functional stenosis at the site of the inferior esophageal constrictor, tertiary contractions of the esophageal musculature, and a pulsion diverticulum.

become almost entirely impossible for a period of two months. An esophagram obtained fourteen years previously at another hospital demonstrated the presence of both a large pulsion diverticulum and a mega-esophagus (Fig. 6, *A*). Esophagrams obtained prior to surgery (Fig. 6, *B*) demonstrated the diverticulum to be so large as to overshadow completely the mega-esophagus radiographically but the markedly dilated esophagus was apparent fluoroscopically.

At the time of the operative dissection (Fig. 7, *A*) a markedly hypertrophied circular muscle band was noted just distal to the stoma of the diverticulum which appeared to be acting as an obstructing agent. Dilatation and tortuosity of the esophagus were present mainly above the stoma of the diverticulum. Because of the esophageal redundancy and dilatation above the

diverticulum, a diverticulectomy and partial esophageal resection with primary esophageal anastomosis was performed. Approximately 5 inches of esophagus above the diverticulum and 1 inch below were resected.

The patient's immediate postoperative course was uneventful. He began eating a soft diet four days postoperatively and was discharged on the seventh postoperative day. His family was enthusiastic about forcing feedings

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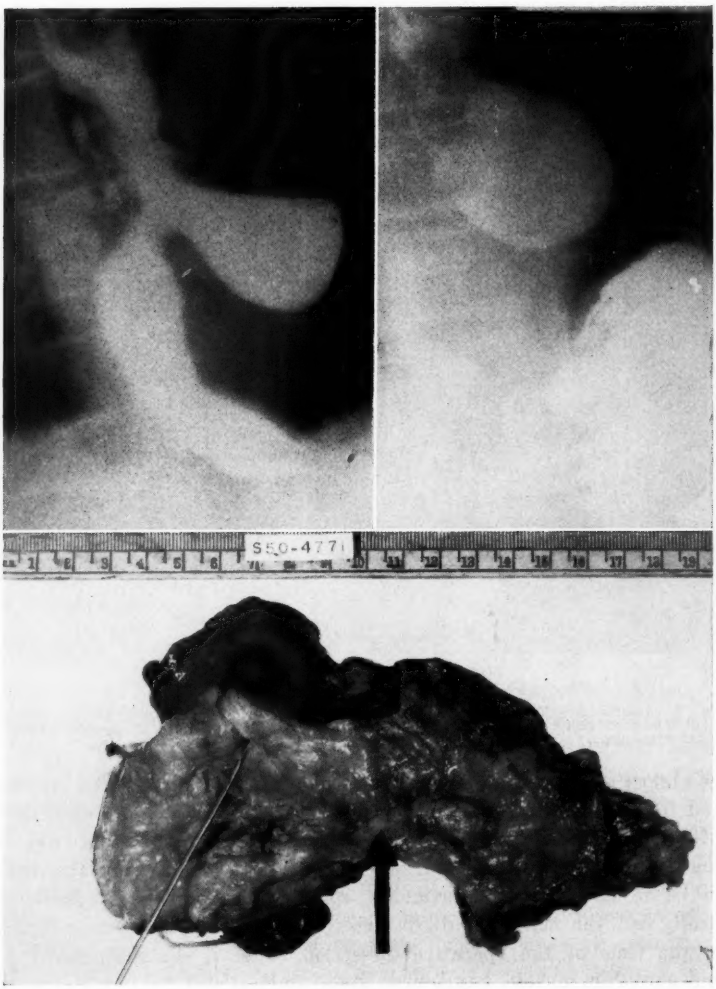
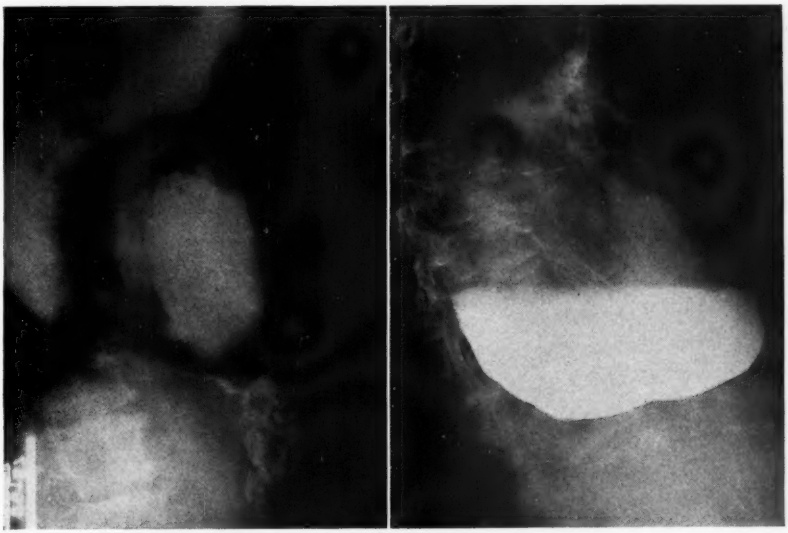


B.

Fig. 5.—*A*, Esophagram on the right demonstrating a functional obstruction to the barium swallow at the site of the inferior esophageal constrictor, a pulsion diverticulum, and a carcinoma of the esophagus. *B*, Surgical specimen demonstrating site (arrow) of bulge into mucous membrane by the inferior esophageal constrictor, the probe in the stoma of the pulsion diverticulum, and the carcinoma of the esophagus.

at home in order for him to gain weight and strength. After four days of this vigorous feeding policy he suddenly developed pain in the right chest and was readmitted to the hospital. He was found to have a right empyema secondary to esophageal perforation. He soon developed cardiac failure and expired. Post-mortem examination of the esophagus revealed a perforation of the suture line. Just distal to the perforation the inner circular muscle was found to be considerably more hypertrophied than that upon the proximal side as demonstrated in Fig. 7, *B*. It was felt that the constricting element of the hypertrophied inner circular muscle in the lower esophagus responsible for both the achalasia and the pulsion diverticulum had been incompletely removed at the time of the esophagectomy and was still causing sufficient functional stenosis to be responsible for the subsequent perforation at the anastomosis.



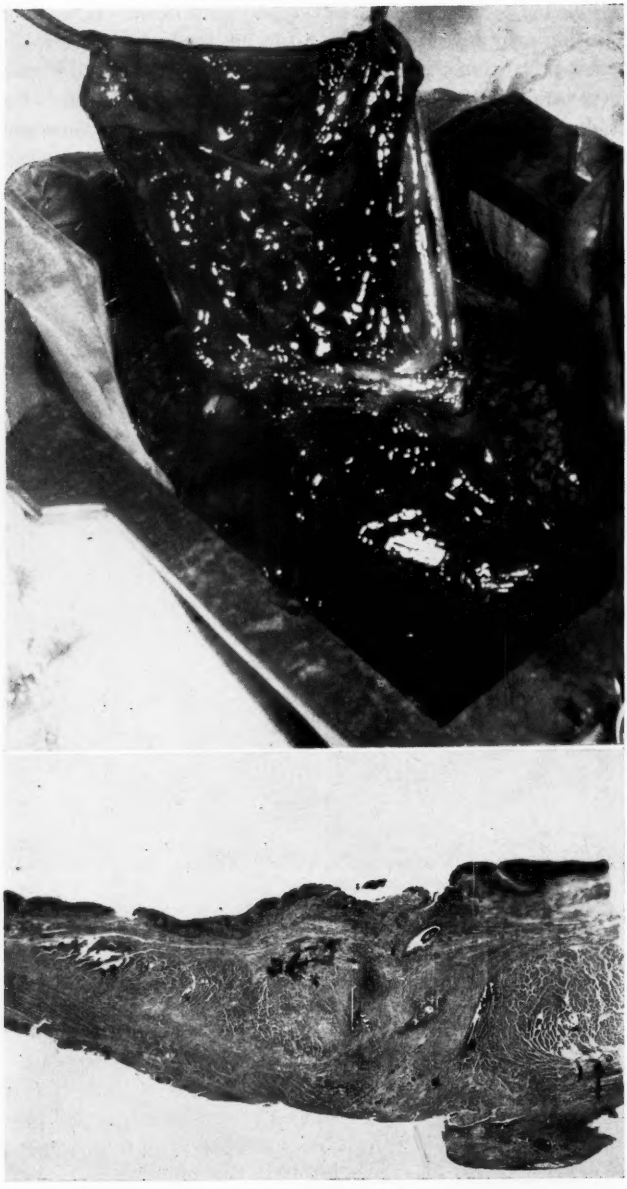
A *B*.

Fig. 6.—*A*, Esophagram demonstrating an achalasia of the esophagus with dilatation except in the terminal esophagus and a large pulsion diverticulum. *B*, Esophagram of the same patient fourteen years later demonstrating increase in size of pulsion diverticulum.

In view of these observations it is felt that functional stenosis in the distal esophagus from a hypertrophied inferior esophageal constrictor causes increased intraesophageal pressure and herniation of the mucous membrane through weak areas in the inner muscle layer to form pulsion diverticula. The cause of the hypertrophied inferior esophageal constrictor is still problematical but probably related to one of the theories predisposing to a preponderance of sympathetic control as discussed previously in this paper.

The next and last group that may have a similar etiology are the strictures in the distal esophagus occurring in the older age group. Three types of patients may or may not fall into this group. The first group includes those patients with hiatal hernias who may have a relaxed or hypotonic inferior

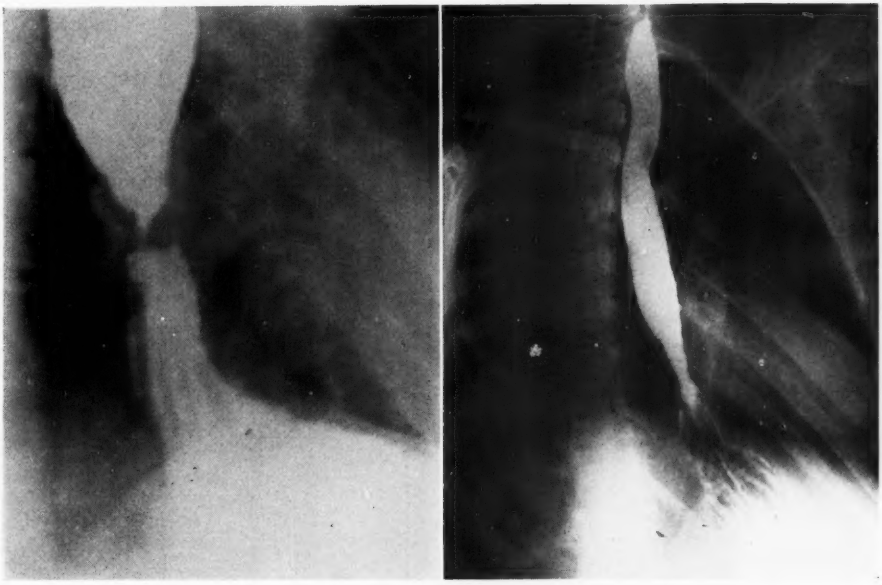
A.



B.

Fig. 7.—*A*, Surgical dissection demonstrating a pulsion diverticulum of the esophagus. *B*, Microscopic section demonstrating hypertrophy of the circular muscle (arrow) at the site of the inferior esophageal constrictor just distal to the esophagoesophageal anastomosis.

esophageal constrictor associated with a relaxed or atrophic diaphragmaticoesophageal ligament. The ascending limb of this ligament arises from the fascia on the undersurface of the diaphragm, extends through the esophageal hiatus, and attaches itself to the longitudinal and circular muscle fibers of the lower esophagus at the site of the inferior esophageal constrictor or sphincter. This ligament is the antagonist of the longitudinal muscle of the esophagus and prevents the esophagus from pulling the stomach into the chest during swallowing.



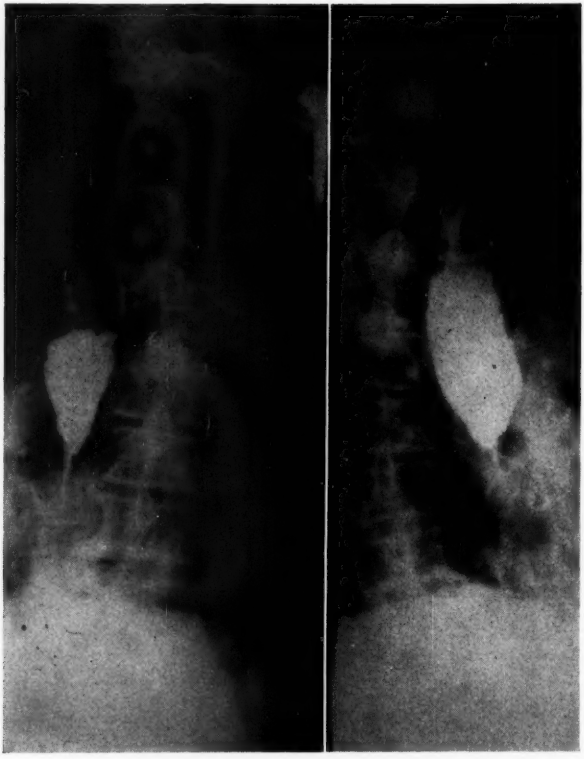
A. *B.*

Fig. 8.—*A*, Esophagram demonstrating an esophageal stricture secondary to a hiatal hernia. The atrophic diaphragmaticoesophageal ligament allows the stomach to herniate into the mediastinum and a hypotonic inferior esophageal contractor allows regurgitation of gastric juice with secondary peptic esophagitis. *B*, Esophagram following treatment by bouginage and ulcer management.

There appears to be an interrelationship between the diaphragmaticoesophageal ligament and the inferior esophageal constrictor in reference to the opening-closing mechanism of the terminal esophagus. Normal individuals can obstruct the flow of barium in the terminal esophagus by taking a deep inspiration at which time the ascending limb of the diaphragmaticoesophageal ligament becomes taut. This can be demonstrated to a greater degree in patients with achalasia. Furthermore the inferior esophageal sphincter in patients with achalasia appears to be hypertrophied.

Opposed to the findings present in achalasia, the relaxed or atrophic diaphragmaticoesophageal ligament present in hiatal hernias allows the stomach to herniate into the posterior mediastinum, and the hypotonic in-

ferior esophageal constrictor allows regurgitation of gastric juice with secondary peptic esophagitis (Fig. 8). If the esophagitis persists, secondary scarring and stricture formation in the terminal esophagus develop. Furthermore, permanent shortening of the esophagus may occur with loss of the sliding action of the hiatal hernia. This syndrome is frequently referred to as a congenital short esophagus with thoracic stomach. The latter condition does occur but its incidence is not as great.



A. *B.*

Fig. 9, *A* and *B*.—Esophagrams demonstrating an idiopathic stricture in a 73-year-old patient treated by resection.

The second type includes those patients with idiopathic strictures of the terminal esophagus without evidence of either a hiatal hernia or congenital short esophagus. This mechanism to a certain degree may again take place in this type, that is, there may be a faulty closing mechanism in the terminal esophagus which allows regurgitation and secondary peptic esophagitis with stricture as just described. These patients usually give a long history of eructation and heartburn suggestive of regurgitation esophagitis prior to the

development of the stricture. Esophagitis can frequently be seen by esophagoscopic examination.

The third type includes those patients who develop difficulty in swallowing and who do not have pre-existent esophageal or gastric complaints. These patients are usually older and have evidence of other degenerative diseases. In such patients it is possible that the organic stricture may be secondary to esophagitis produced by stasis resulting from functional obstruction by a hypertrophied inferior esophageal constrictor (Fig. 9). Most of these patients can be relieved by medical management including an ulcer diet, other buffers, and esophageal dilatation so that further observations from surgical dissections are lacking. The periesophageal fibrosis and mural scarring present in those patients requiring surgical resection of the stricture mask any possible etiological factors.

SUMMARY

Observations are presented which suggest that either spasm or hypertrophy of the inferior esophageal constrictor may cause functional stenosis and be a factor in the production of functional dysphagia, the Plummer-Vinson syndrome, esophagospasm, achalasia, pulsion diverticula of the thoracic esophagus, and idiopathic strictures of the terminal esophagus. The etiology of this spasm or hypertrophy is not clear but undoubtedly is similar to spasm of circular muscle in other smooth muscle sphincters in the body. There is both experimental and clinical evidence to suggest that this spasm and hypertrophy result from a preponderance of sympathetic control. Possible causation of this sympathetic imbalance is discussed.

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DISCUSSION

DR. I. DARIN PUPPEL, Columbus, Ohio.—I think we should all congratulate Dr. Kay for his contribution to the understanding of achalasia and other diseases of the lower end of the esophagus.

Certainly there is still much lacking in our knowledge of the etiology and the treatment of this group of diseases, particularly achalasia. We know that normally esophageal swallowing depends to a great extent upon peristaltic movements which expel the intraluminal contents of the esophagus into the stomach. At least those of us who have lived on farms know that in childhood we were able to drink uphill from a spring of water. This upward esophageal swallowing was dependent almost entirely upon peristaltic movement of the esophageal musculature. Hurst, in his important studies and papers over a period of about two decades in the early part of this century, emphasized that the achalasia disease was

localized to the cardiac end of the esophagus. It was common opinion that peristalsis occurred normally in mega-esophagus but that it was ineffective in deglutition because the bolus of food was not large enough to be grasped by the hugely dilated esophagus. However, in recent years, many investigators have been convinced that this disease is much more generalized, involving much more of the esophagus than was heretofore realized. We, for instance, published a paper in the JOURNAL OF THORACIC SURGERY a few years ago, in which we emphasized that in four cases of achalasia there was a definite involvement of the whole esophagus in functional disturbances. Not only the smooth but also the striated musculature of the esophagus was involved in these disturbances. These physiologic abnormalities were not necessarily greatly changed by relief of the obstruction at the cardia. Using inflated balloons at various levels of the esophagus which acted as boluses of food to stimulate the esophagus to movement, and recording the movements on a long kymograph, we found the intrinsic motility of the whole neuromuscular esophagus in achalasia to be irregular, uncoordinated, hyporesponsive to stimuli, and never completely effective. In fact, the intrinsic motility was completely absent in some cases. Kramer and Inglefinger, of Boston, are the only men to our knowledge who have done similar studies. It was interesting that they, publishing their work almost one year after we had submitted our paper for publication in the JOURNAL, independently confirmed that portion of our research in which they were interested.

We now have similarly studied eight additional cases of achalasia. We know from these experiences that every so-called idiopathic mega-esophagus shows a profound change of varying degree of the intrinsic motility with little or no production of peristalsis and great decrease in the power of deglutition. Usually in the fusiform type we found slight disturbances, in the flask type moderate disturbances, and in the sigmoid or zig-zag type, which we feel is the far-advanced type of mega-esophagus, we have found none or little intrinsic motility. Barrett has recently written that he has never seen the fusiform transform into the sigmoid type of mega-esophagus. Yet I have available roentgenograms of barium swallow studies of one patient over a period of fourteen years. These revealed a fusiform mega-esophagus in 1937 which transformed to a sigmoid type by 1951. I did an esophagogastrostomy, bilateral vagotomy, and a posterior gastrojejunostomy for this patient and she is now well.

In two cases of uncomplicated supradiaphragmatic diverticula of the esophagus, as proved on excision at surgery, we were not able to elicit preoperatively or postoperatively disturbances of the esophagus proximal to the diverticulum. We feel that the two diseases, physiopathologically speaking, are different in nature.

When we administered a sympathicolytic drug such as Priscoline or a parasympathomimetic drug such as Urecoline alone or in combination, we found that the mega-esophagus lumen usually reduced in size and some increase in the rate of emptying of the esophageal contents occurred. Tracing studies showed frequently marked increase in tonus, frequency, and power of contractures of the esophageal musculature. And yet these drugs were disappointing in their therapeutic value for the reason that almost always the vigorous movements still remained localized, pathologic, uncoordinated, and nonperistaltic in nature. They merely led to an increased kneading or churning effect so that the power of esophageal deglutition was usually not appreciably enhanced, thus differing from the effects of these drugs on the hypomotility of the atropinized normal esophagus.

Our data indicate there is a marked disturbance of the physiology of the whole esophageal neuromusculature. They indicate that our present modes of treatment cannot entirely relieve the abnormal physiology and that other better concepts will have to be sought. All present methods of treatment have their disadvantages and failures. We now routinely do preoperative overnight twelve-hour gastric secretion and histamine gastric analysis studies. For those patients who show free hydrochloric acid we have used Banthine, and have done bilateral vagotomy together with a gastrojejunostomy or a partial gastric sympathectomy besides relieving the esophagogastric obstruction, and have noted remarkable improvement in end results in certain cases. Esophagitis did not occur in two cases maintained achlorhydric postoperatively. Achalasia of the esophagus is a progressive disease and, on the whole, better therapeutic end results may be expected if the diagnosis is made early and treatment is instituted early in the course of this disease.

DR. J. KARL POPPE, Portland, Ore.—I would like to take this opportunity to mention one method of treating epiphrenic diverticula which should not be used. I refer to my own experience with two cases of large epiphrenic diverticula associated with esophagitis, diverticulitis, stricture, and spasm of the lower esophagus and cardia. In each of these two patients a very simple method was used, anastomosing the fundus of the stomach to the bottom of the diverticulum, a relatively simple procedure which can be done readily, relieving the diverticulitis. This anastomosis worked well for a period of approximately eight months in one case, at which time the suture line apparently opened and a fistula occurred between the diverticulum and the pericardial sac. We drained that by a large pericardiotomy and the food came out through the pericardiotomy opening in the anterior chest for a few weeks. The fistula gradually closed by an obliterative pericarditis. The patient unfortunately expired from an infarction in the right lung a few weeks later.

The other patient did much better than that. Swallowing was satisfactory for about three years, at which time he also developed a fistula and began to cough up food. I understand this required a lobectomy and resection of the lower esophagus. A radical resection of the lower esophagus including the diverticulum and stricture should have been done in the first place to remove the infected diverticulum and obstruction of the lower esophagus in each case.

DR. J. L. EHRENHAFT, Iowa City.—We have recently been interested in this particular problem, and at the present time there are functional motion studies being carried on by Dr. Taber in our hospital which are rather interesting. Up to the present time all the studies have been done by single balloons placed in the esophagus. Dr. Taber has inserted three balloons at different levels, hypopharynx, mid-esophagus, and lower esophagus, which are swallowed, and they are connected simultaneously to very sensitive kymographs. We have studied about six or seven patients with achalasia up to the present time. This being a functional disease which is certainly progressive, we see these patients quite late. The tone and the peristalsis of the muscles are greatly decreased. We recently had a patient who was being treated for another condition, whom we used as a control for a normal esophagus. At the time the balloons were swallowed and the motion studies were done, there was complete absence of contraction of the esophageal musculature. Esophagrams showed a dilated esophagus with cardiospasm.

EXPERIENCES WITH THE OPERATIVE MANAGEMENT OF DELAYED RESTORATION OF ALIMENTARY CONTINUITY IN CHILDREN ORIGINALLY TREATED BY THE MULTIPLE STAGE PROCEDURE FOR CORRECTION OF CONGENITAL TRACHEOESOPHAGEAL DEFECTS

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St. Paul, Minn.

INTRODUCTION

AMONG most surgeons who deal with the congenital tracheoesophageal fistula problem there currently exists a clearly understandable sentiment for salvaging the greatest possible number of children. To those deriving guidance from this principle, it is apparent that the routine insistence on the use of a primary anastomosis will inevitably yield a smaller percentage of successes. Contrariwise, an opportune use of the multiple stage procedure, in the instances of simple atresia, will often save the life of one more child. Regardless of one's scientific or emotional bent, and we personally strongly oppose an "all-or-none" operative philosophy for the management of this grave ailment, an occasional need will surely arise for techniques of bridging a substantial segment of the esophagus. Because of these considerations we are reporting our experiences with the resolution of this situation in ten children originally treated by the multiple procedure technique of fistula ligation, cervical esophagostomy, and gastrostomy.

HISTORICAL

In an earlier presentation with Lannin and Tongen[12] we reviewed the items of the embryologic variety of lesions occurring in congenital atresia of the esophagus, the historical attempts at therapy for this problem, and our experiences with 103 consecutive cases,* and considered in some detail the general and the specific surgical care for this situation. From this group, there were ten children originally treated by the multiple stage procedure, on whom we operated for restoration of alimentary continuity.

During the years intervening since Leven and, independently, Ladd achieved success with the multiple stage technique, numerous surgeons have skirmished with this problem in reconstruction. Some have sought to develop an antethoracic skin tube. Ladd,[8] Ivy,[7] and Lam[11] did employ some variant of this plan for completing intestinal continuity in the tracheoesophageal fistula cases; Davis and Stafford,[3] Stevenson,[21] and Hanrahan[5] developed skin-lined simulated gullets for comparable situations. Though completed tunnels were

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*This includes sixty-eight instances of primary anastomosis with forty-three survivors.

reported, early and late technical tribulations were met. Closure difficulties arose from primary or recurrent fistula formation as well as from strictures eventuating particularly at the gastrocutaneous anastomotic junction. Ochsner and Owens,[16] Lam,[10] and Ladd and Swenson[9] sought to reduce the item of scarring from acid peptic juice by bringing to the anterior abdominal wall the distal esophageal segment.

The effectiveness of this maneuver in reflux control is questionable, however, since the dissection required would seriously compromise one's normal esophagogastric pinchcock mechanism. These several limitations contributed to a rather tepid enthusiasm among many surgeons for this general management program and did encourage the planning of alternative approaches.

Yudin[25] has described in some detail many techniques, essentially of historical interest and but infrequently attempted currently save for that procedure pioneered by César Roux. The use of a limb of jejunum, secured from the segment just distal to the Treitz ligament, as an antethoracic esophageal replacement, received the major attention of Yudin. In fact, the Y-type anastomosis seemed to represent his preferred method for bridging long caustic or acid-induced esophageal strictures. His description and suggestions for the technical management of the mesenteric dissection served reliably for guidance while we, from a quite inexperienced position, were acquiring some familiarity with this problem. A careful study of this paper is recommended for those planning to use the jejunum for thoracic esophageal replacement. Despite Yudin's favorable support of the method, the development of a length of vascular mesentery so adequate and uncompromised for viability as to permit a safe union with the cervical esophagostomy was at times a poser and led to its virtual abandonment by some.[4] A clever and singular success in meeting this issue was described by Longmire[14] who reanastomosed one set of the vasa recta jejunalis to the internal mammary vessels. Longmire and Ravitch[13] effectively developed a parasitic blood supply from the surrounding pedicle tube for a length of transposed jejunum by a careful staging through several steps so that ultimately complete division of the mesenteric tributaries was achieved.

Van Prohaska and Sloan[23] sought to realize a greater transplantable length in the jejunal segment by denuding the mucosa of the serosal and muscular layers. The remaining free, full thickness, mucosal tubes were rather uniformly nonviable in their canine experiments, and use of this technique was followed by some stenosis in one clinical counterpart. Harrison,[6] as well as Robertson and Sarjeant,[19] shortened the distance to be bridged by recourse to a more direct routing of the jejunal limb between cervical gullet and stomach. Rather than the usual arclike position described by a subcutaneously placed antethoracic intestinal segment, the bowel was carried more like a secant directly through the thorax by the former and just substernally by the latter team. Cosmetically, these methods revealed certain advantages, as well as minimizing any likelihood of complications secondary to severe compressive trauma against the firm chest wall. On the other hand, to all persons cognizant of children's dietary indiscretions, the accidental impaction of some unusual sized particulate bolus within

this intestinal segment is a possibility and might likely be at a quite inaccessible site. Endoscopic instrumentation through the kinked bowel segments, too, would be hazardous.*

Rienhoff,[18] Bigger,[1] and Potts[17] have mobilized the stomach to complete a delayed antethoracic esophagogastric anastomosis. The latter author subsequently placed these segments within the left thoracic space after division of the appropriate costal cartilages.

Sweet[22] and Clifton,[2] in a delayed reconstruction of alimentary discontinuity secondary to esophageal atresia, moved the stomach into the chest and out the superior thoracic aperture. A gastric anastomosis with the cervical esophagus was completed in two cases recorded by the former. Food consumption was described as essentially normal, though growth came a bit slowly to one child among Sweet's cases. During subsequent years this child improved at a more rapid pace. No untoward physiologic disturbances were noted or ascribable to the intrathoracic stomach. In a subsequent publication, Longmire[15] carried through a reconstruction plan resembling Yudin's, in that a jejunal segment pedicled on a single vertical mesenteric tributary was mobilized into an antethoracic position. By this technique, alimentary continuity was restored to two infants shortly after thoracic exploration revealed the nonanastomosable hypoplastic lower esophageal segment in association with a tracheoesophageal fistula. Their subsequent physical development and health as recorded in a follow-up at twenty-eight and thirty-nine months, respectively, has been gratifying. Delayed sectioning of alternate principal elements of the vascular rete has been studied by Shumacker.[20] For the rat and for the dog, by this means, a greater length of intestine could safely be pedicled. Its successful application to a clinical case of lye stricture in a child was described.

Our own first cautious groping for some acceptable solution to this situation began in 1947. For minimum standards we desired a technique surgically safe, compatible with a reasonable rate of physical development, and one which would be unlikely to prejudice the future should an anatomic rearrangement at a later date prove essential. It should also have enough cosmetic appeal to prevent rejection or scornful comparisons when this child would be exposed to the comparative glances and comments of his playmates. The reconstructions in nine of the ten cases being reported seem to meet these requirements reasonably well.

METHOD

As a general plan these children were admitted a few days before the first stage of the reconstructive procedure. During these twenty-four hours immediately prior to surgery, at least two intramuscular penicillin injections were given; streptomycin or Aureomycin was supplied via the gastrostomy, but no nutrient was permitted by this route during the last twelve preoperative hours. The anesthetic for each stage in virtually every operation was Pentothal Sodium and curare, for induction and for maintenance. A high concentration of oxygen in combination with nitrous oxide was supplied through an endotracheal tube. These anesthesias, administered by a large and changing number of the medical personnel in training for that specialty, were invariably superbly well done. Midline, right or left upper

*In one of our cases, a swallowed penny became so inextricably wedged into the esophageal pouch as to require transcervical removal.

rectus, or transverse incisions all offered an adequate exposure. The incision of preference of the original operator at the time the abdomen had been entered to make the gastrostomy was reopened to develop the exposure about the area of the jejunal mesentery. Any and all adhesions to the upper small bowel and its mesentery were first freed. The sheaths of peritoneum were preserved over all arcuate vessels immediately next to the bowel. It was cleared away, however, along with all fat and often a considerable quantity of variously sized and clustered lymph nodes, directly down to the first portion of the main superior mesenteric vessel to the entire small bowel. This arterial segment and the first four or five major primary channels were carefully dissected free of the network of sympathetic fibers ramifying locally and distally. The vasa recta jejunalis to be responsible for nourishing the transplant and the superior mesenteric artery at this level was denuded of autonomic fibers and areolar substance quite as carefully as one prepares a small artery for an anastomosis. Vasospasm was not sustained in these periarteriectomized segments. With serrefine clamps applied to as many as three or four of these main tributaries, reflections from pulsations were usually visible along the vascular arcades immediately adjacent to the moist bowel. The first large vascular channel to the jejunum was always saved to insure a sound blood supply on the proximal line of the intestinal anastomosis. With the bowel sectioned 6 to 10 cm. distal to the Treitz ligament it as periodically tested for adequacy of length by measuring it onto the anterior abdominal and chest wall. Redivision of the straight vessels and then occasionally those of the first arcade served to straighten the intestinal length relative to mesenteric length. After experiences with uniting the upper end of this jejunal segment to the esophagus, once or twice, it was self-evident that enough redundant bowel to bridge any reasonable gap could be dissected free at that time. Any all out effort to bring this jejunal limb precisely to the cervical stoma was therefore unnecessary and might even compromise its terminal few centimeters. After developing an approximately adequate length of this prepared portion of bowel, it was passed through a rent in the transverse colonic mesentery and then brought out from the abdomen in a midline fascial opening. It then dipped into a widely undercut subcutaneous tunnel, finally popping out through a loosely fitting skin incision at about the level of the clavicle or first rib. To minimize tension in this antethoracic tunnel the area of the chest and abdominal wall was widely freed over an area from the right side of the sternum to the left anterior axillary line. Serosanguineous accumulations in this raw region were led away by two or three laterally placed soft Penrose type rubber drains coming out of stab incisions. Intestinal continuity was re-established by end-to-side union (Roux Y) below (retrocolic) the transverse colonic mesentery. This tissue was tacked to the loop of jejunum and then the abdominal fascia was sewn to the bowel as it left the peritoneal cavity—quite similar to the technique for preventing herniation about an ileostomy. Attention was ever directed against the development of mesenteric venous engorgement in the transplanted loop either through compression or twisting, or from inadvertently placed sutures. The short external segment of bowel was left open to preclude the serious accumulation of internal tension from secretions. It usually became cyanotic in varying degrees. Its viability was tested and deemed all right in the first patient by fluorescence to ultraviolet light after an intravenous injection of fluorescein. Continuity in the cervical region was accomplished at intervals of a few days to several months following the development of the antethoracic jejunal segment. In the first cases, this delay was deliberate in order to permit the edema and other acute inflammatory reactions, aroused by the first procedure, to subside, so that the bowel could then be more easily sewn to the esophagus. The likelihood of a salivary fistula of even a few days' duration is probably lessened by a reasonable exhibition of patience. These anastomoses were all of the open type, two rows, with the inner one mucosa-to-mucosa union, and were completed with 00000 silk.

Once we recognized the essential need for correcting the "by-pass" of the stomach by the jejunal segment, another stage was added to the reconstruction. The abdomen was opened once more and the jejunal limb of the Y anastomosis divided close to the duodenum, leaving a small blind pocket. The excess bowel was now resected after devascularizing it close to the mesenteric border. Several kinks thereby could usually be removed. This straightened segment was then joined to the antrum. Most of the feeding gastrostomies were

maintained for several months to supplement the child's caloric intake. When eventually abandoned they rarely healed spontaneously, but required a coring out of the tract plus a formal closure of that area.

In one child, J. N., intestinal continuity was established by transplanting the entire stomach into the left thorax. This dissection was carried out through a thoracoabdominal incision. An esophagogastric anastomosis was made in the neck after an extraperiosteal removal of the first rib provided an ample-sized opening to deliver into the cervical area the gastric fundus.

RESULTS AND DISCUSSION

To date the reconstructions have been completed in eight children through the stages of esophagojejunostomy, jejunogastrostomy, and closure of the gastrostomy. Their ages, current heights, weights, and dates of last operation are listed in Table I. One boy has only the cervical esophagus anastomosed to the jejunal limb; another child has had alimentary continuity established by esophagogastrostomy after transplantation of the stomach into the left thorax and lower neck. The first reconstruction was started as a jejunal transplant in a 3½-year-old boy on June 5, 1947. On Aug. 21, 1947, the upper stoma was united to the esophagostomy and convalescence was uneventful. At times, and unpredictably in relation to the kind of meal eaten, this child has had some of the manifestations of a "dumping" syndrome, that is, tachycardia, sweating, and pallor. He would often then express a desire to lie down. Nausea was not noticeable. There was a slow but steady gain in weight and appetite evidenced by the youngster. Because of his generally acceptable over-all status the need for transplanting the jejunum back onto the stomach was not at once apparent.

TABLE I. POSTRECONSTRUCTION GROWTH RECORD FOR ALL CHILDREN AS OF MAY 1, 1952

PATIENT	AGE (YR.)	CURRENT WEIGHT (POUNDS)	CURRENT HEIGHT (INCHES)	DATE RECONSTRUCTION COMPLETED
D. E.	9			4/29/49
D. W.	8½	55	51	9/ 9/49
D. G.	9	66¼	54½	10/25/49
O. B.	6½	51½	47	11/15/49
J. B.	8	51½	46½	12/30/49
J. N.	7	33¾	45	1/20/50
R. L.	12	80	56½	3/31/51
G. B.	6½	35	43	6/ 6/51
M. S.	5	37	42	7/24/51
D. S.	5	40½	43	9/28/51

Our second patient, anastomosed for oral alimentation Sept. 16, 1948, did so poorly in the following few months (alopecia areata, little weight gain, reluctance to eat, and frank personality changes) that a jejunogastrostomy was performed in April, 1949. Subsequent to this procedure the child improved steadily and he has maintained a fair growth rate since. In other cases of this series we have noted the progressive improvement in the individual's general health once the stomach has been restored to the direct line of alimentary continuity. Longmire has recently commented upon the importance of not "by-passing" this organ. (Yudin's statement to the contrary might be accounted for by the older age of the patients in his series, and hence their lowered

metabolic rate.) For children, we believe there are real advantages to having it connected directly into the food circuit. The antrum was selected in all cases for union with the jejunum. The anastomosis was deliberately placed here recognizing that should a stomal ulcer ever trouble the individual later in life, it would still be possible to resect, transthoracically, enough of the acid-bearing area to cure the ulcer diathesis, without disturbing those already existing anastomoses. Theoretically and from experimental evidence there is a somewhat greater likelihood of an ulceration occurring in the situation where the stomach is "by-passed" than if continuity is made with the jejunum.

The present weight and height of each child in this group have been plotted on a Wetzel grid.[24] Seven of the nine individuals in this group cared for by the jejunal transplant were above or around the 67 per cent auxodrome with three of these sustaining physiques and developmental levels to the 15 per cent auxodrome. When these items of weight, height, and age were plotted for the child reconstructed by an esophagogastrostomy technique, the curve derived was barely on the grid, and far below the 98 per cent auxodrome. In the twenty-seven elapsed months since completion of his anastomosis, this youngster has remained fragile and "puny." Therefore, the likelihood is strong, particularly since he has fallen away from his isodevelopmental line, that this case represents one with a poor physical fitness record. Clinically, there is support for this type of representation. Whereas eating seems to provoke in him no acknowledged distress, exposure to and selection from a variety of nutritious items at any meal rarely captivates his appetite. Roentgenologically the stomach appears flaccid, empties slowly, but ultimately, and shows no organic site of obstruction. It is conceivable that, for this child, vagotomy with its secondary inhibitory effects on pancreatic function has been majorly responsible for a substantial measure of the obvious physical retardation. Obversely, most of the children with a jejunal reconstruction including an anastomosis onto the stomach have done well, and several have sustained an excellent physical fitness record.

The youngest child in this series was 2 years, 9 months of age at the time a jejunal limb was positioned onto the chest wall. The remainder were several months to years older before a surgical reconstruction was attempted. This uncertainty and admittedly timorous viewpoint about beginning the necessary reparative steps on very young children was fostered by an often expressed desire from the parents to await a "best" operation. Several were participants in a close network of correspondents composed of parents raising similarly crippled children. Longmire's excellent results in restoration of oral alimentation with two neonatal infants offer a sound argument in favor of an earlier intervention than we practiced.

Although these youngsters uniformly came to the initial operation for restoration of alimentary continuity in good physical condition, the burden of preparing and patiently injecting thousands of gastrostomy feedings, and then diapering the cervical esophagostomy, was assuredly an enormous labor of love. In this regard some parents had been farsighted enough to encourage their

child to eat and drink from a wide selection of foodstuffs and beverages. This painstaking cultivation of youthful taste buds and palate was rewarded by a more eager acceptance of regular childhood fare once intestinal continuity was achieved. The adjustment, in mouths tender from no contact with the coarser foods, was slower.

The cosmetic result is not nearly perfect in any single case, at least to our critical gaze. Fortunately, the parents have been more charitable, and realistic. There has been no ardent acceptance of our offer to improve the child's appearance by placing the at times prominent intestinal loops beneath the thoracic frame. Of course, this suggestion has always been coupled with an explanation that this procedure could do nothing to improve intestinal function. We have also speculated about the hazard from trauma to the bowel or to its blood supply, by a crushing injury applied to the chest or costal margin. Specifically in boys this potential has provoked some concern for the future. True, the follow-up time has been short, and these children with but a single exception are rather too young for the hardier athletic contests. Still, for this oldest boy, roughhousing, wrestling, and back yard football of a somewhat rugged character have provoked no distressing symptoms nor daunted his zest for such exercise. Some parents are yet considering this final transplantation plan, but most of them are outspokenly grateful now that their son or daughter can eat contently and from the family table. With a greater experience in treating the arcades and vertical nutrient vessels, straighter, less redundant, segments of interposed jejunum have been left. Also, the excess intestine can be tailored anew and shortened at the time of completing the gastrojejunal union. This is one small argument in favor of delaying that stage until a later date in the over-all restoration program. As noted often in the whole series, during fluoroscopic examination of this segment, the main bolus of barium drops under gravity's influence and is pushed by peristalsis through to the gastric pouch, seconds after its oral ingestion. An inability to empty this intestinal limb, granted the meal is not a particularly dry one, has not arisen as a handicap to the acceptance of a good-sized repast in the course of one sitting. A small serous or salivary fistula was noted at the upper anastomosis in three cases. All healing was spontaneous, with the child remaining on clear liquids, in the course of three to five days. The remainder of the cervical esophagojejunal junctions healed primarily without evidence of gross leak, as did all of the intra-abdominal suture lines. In the tenth and last patient operated upon, a slough developed at the superior end about twenty-four hours after the surgery for the first stage. Prior to this time the color in this segment had seemed quite satisfactory. Promptly after the necrotic end was recognized the subcutaneous tunnel was opened under local anesthesia at mid-chest and all nonviable or dubiously viable bowel cut free. Fortunately, an adequate length yet existed to permit approximation of the remaining segment to the cervical esophagostomy at a later date. In this boy the jejunal limb is now straighter than it is in some of the other children, and it functions smoothly.

SUMMARY

Experiences with ten patients in the operative management of delayed restoration of alimentary continuity in children originally treated by the multiple stage procedure for congenital tracheoesophageal defects have been reported. The shortcomings of both methods tried, as well as their advantages, have been noted. The functional results in those cared for by the interposition of a jejunal limb between the cervical esophagus and the gastric pouch were appraised as superior. The single child with stomach united to the upper gullet has failed to mature physically at a standard and acceptable pace. The relevancy and need for a sustained follow-up in this group and in comparable cases, through their later formative years to test the effect of these stresses, as well as aging, are candidly conceded.

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DISCUSSION

DR. FREDERICK G. KERGIN, Toronto.—I am very interested in this presentation by Dr. Varco. Dr. Robert Mustard (of Toronto) and I have also had some thoughts about reconstruction of the esophagus for slightly different types of lesions. We do not do work with children and have not had this exact problem, but we have had young patients who had lost the use of the esophagus, in whom we wished to establish an alternate route for food. We have been very concerned about by-passing the stomach or about anastomosing the jejunum or any other bowel to the stomach because of peptic ulceration. It seemed to us ideal if we could design a technique to put the new esophagus in the chest and anastomose the lower end to the lower end of the esophagus, if the obstruction were above that level, so as to protect the bowel by normal sphincter action of the lower end of the esophagus. We had one such case in which this procedure was successful. We found that the jejunum had too precarious a blood supply for this maneuver, and we used the colon.

Our patient was a young woman who had a paraffinoma of the mediastinum, secondary to an old extrapleural pneumothorax with paraffin oil fill. She had intractable stenosis of the upper esophagus. We were able to mobilize an isoperistaltic loop of transverse colon pedicled on the middle colic artery, pass it up through the left chest and into the neck, and anastomose the upper end to the upper end of the esophagus, and the lower end to the esophagus just above the diaphragm, so as to by-pass the upper esophagus. She has done well and now has excellent function. I think in cases of this type, if there is a lower segment of esophagus available, which is functional, it is perhaps more ideal from the cosmetic as well as the physiologic point of view to put the new esophagus in the chest and preserve the esophageal sphincter.

DR. MARK RAVITCH, Baltimore.—The problem of peptic ulceration in jejunal loops that have been anastomosed to the stomach is not purely a theoretic one. We have observed this twice, both times in young women who have had an antethoracic esophagoplasty performed for a lye structure. On the other hand we have twice had infants, such as Dr. Varco presented today in his interesting paper, who failed to gain weight and had intractable diarrhea, the stomach having been by-passed, and who did very well and continued to do well after the lower end of the antethoracic jejunal loop was imp'anted into the stomach. It is Yudin's practice to by-pass the stomach routinely. We recently had a visitor from Yugoslavia who, during the Yugoslavian-Soviet honeymoon, spent several years with Yudin, and who has a very large experience of his own in Belgrade—Dr. Papo. His practice is the same, to by-pass the stomach—these are mostly lye and sulfuric acid strictures—anastomosing the loop to the stomach only if diarrhea or malnutrition results, and apparently it can result, even in adults.

One final historical note about Roux's case. That case was reported again forty odd years later by his original first assistant who had taken Roux's last name, possibly having

married Roux's daughter (I have not checked on that) and he is now Picot-Roux. He points out, in a French article, that not only had the patient progressed very well but he had sired two fine children.

One last technical point which is brought to mind by Dr. Kergin's reference to the use of isoperistaltic loops. There are at least two references in the literature to the advantage of reversing the jejunal loop in order to take advantage of the regularly greater size of the first jejunal artery. A careful reading of those papers would indicate that the patients have all succumbed. There is an old reference to Jankouskis of Riga which is carried down in modern literature, and again careful reading of his article shows, in fact, that none of the loops were antiperistaltic, they were all isoperistaltic.

SURGICAL TREATMENT FOR CONGENITAL AORTICOPULMONARY FISTULA

Experimental and Clinical Aspects

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David C. Sabiston, Jr., M.D. (by invitation)
Baltimore, Md.

A DEVELOPMENTAL defect in the aortic septum with communication between the aorta and the pulmonary artery near the base of the heart is a rare congenital anomaly. Gasul, Fell, and Casas,[1] who reported a case of this type in 1951, were able to find only twenty-four other cases recorded in medical reports during the last century. Little is known about the origin of the anomaly except that it probably results from an arrest in the development of the aortic septum between the fifth and eighth weeks of intrauterine life.[2] In the normal fetus the growth of the aortic septum divides the primitive truncus arteriosus into two separate vessels, the aorta and the pulmonary artery. Failure of growth or of fusion of the anlagen of the septum may result in persistence of certain features of the truncus arteriosus in postnatal life. Depending on the degree of the arrest, the windowlike communication between the aorta and the pulmonary artery which results may vary from a small round opening 5 mm. or so in size located 8 or 10 mm. above the cusps of the pulmonary valve to a large defect which extends from the valve cusps to the bifurcation of the main pulmonary artery (Fig. 1).

Congenital aorticopulmonary fistula is probably the best descriptive term for this anomaly from both the physiologic and clinical points of view. The alterations in circulatory dynamics associated with it appear to be similar to those produced by persistent patency of the ductus arteriosus. The clinical syndrome has been discussed recently by Spencer,[3] Gibson,[4] Gasul,[1] Dammon,[5] and their respective associates.

Our interest has been primarily concerned with the development of a method of surgical treatment for congenital aorticopulmonary fistula. The purpose of this article is to describe experimental techniques for production and closure of aorticopulmonary fistula and to report the successful clinical application of the method of closure in an infant.

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EXPERIMENTAL ASPECTS

Production of Aorticopulmonary Fistulas.—The object of these experiments was to produce a laboratory preparation which would resemble anatomically and physiologically congenital aorticopulmonary fistula in patients. Several years ago during the development of the technique of creation of an artificial ductus arteriosus, Dr. Alfred Blalock devised a means of producing an aorticopulmonary fistula which consisted of placing a stab wound between the pulmonary artery and the aorta near the base of the heart where the walls of the two vessels are in apposition. This technique did not prove to be entirely satisfactory, however, and it was found by Blalock and his co-workers that fistulas produced in this way tended to close spontaneously.[6]

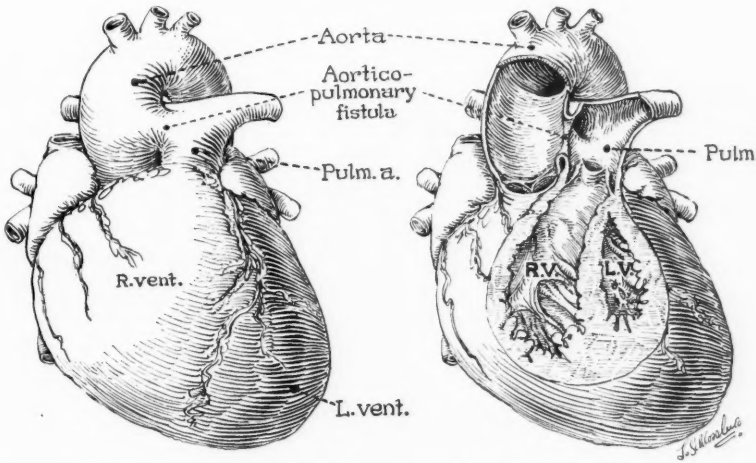

Fig. 1.—Sketch of heart at autopsy on 18-month-old baby with congenital aorticopulmonary fistula exhibiting typical anatomic features of the anomaly.

The method adopted for use in the present experiments is the construction of a suture anastomosis between the ascending aorta and the main pulmonary artery. It is essentially a modification of the technique of lateral anastomosis between the descending aorta and the left pulmonary artery described by Potts, Smith, and Gibson.[7]

Twenty-five adult mongrel dogs were used in these experiments. The procedure which was employed in the production of aorticopulmonary fistula in these animals was as follows. After morphine and atropine premedication, the animal was anesthetized with ether. Pulmonary ventilation was maintained by the use of an intratracheal tube and a mechanical insufflator.[8] Using aseptic technique an incision was made in the fourth left intercostal space. The pericardium was opened widely. The intrapericardial fat pad overlying the base of the aorta and the pulmonary artery was excised and both vessels were carefully freed of investing areolar tissue down to their points of origin

from the heart. With the vessels widely mobilized, a curved Potts ductus clamp was applied to the anteroinferior aspect of the ascending aorta so as to isolate a crescentic segment of the aortic wall. A similar segment of the wall of the adjacent main pulmonary artery was isolated by the application of a straight Potts ductus clamp to its anterosuperior aspect. Each clamp was applied in parallel to the long axis of the vessel. A small incision was then placed in the wall of each isolated vascular segment and a lateral anastomosis constructed with 00000 arterial silk sutures by the Blalock method.[9] After the removal of the clamps a thrill was palpable in the pulmonary artery. The pericardium was closed loosely, the lung re-expanded, and the chest wall closed in layers. The steps in technique are illustrated in Fig. 2.

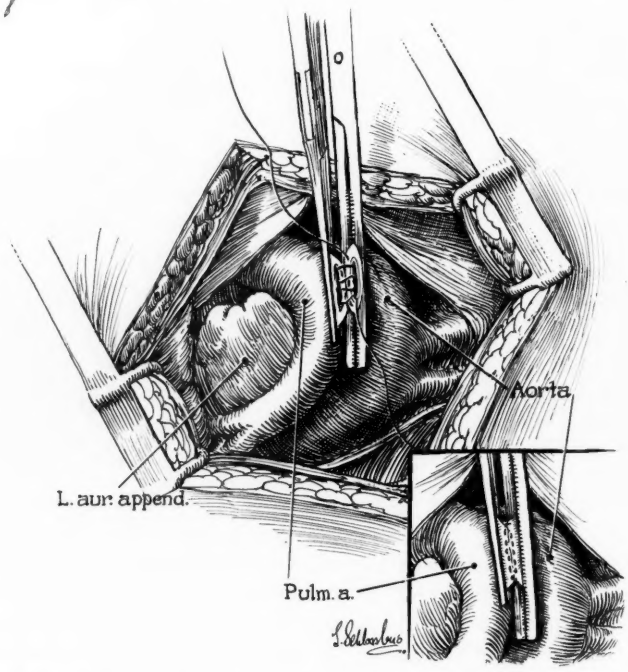

Fig. 2.—Technique of production of experimental aorticopulmonary fistula.

With this method it is possible to place the anastomosis within one centimeter or less of the aortic valves and even closer to the pulmonary valves. A typical example of a small fistula produced by this technique is seen in Fig. 3.

Three of the twenty-five dogs used in these experiments died of hemorrhage from the anastomosis in the immediate postoperative period. In each of the remaining dogs an aorticopulmonary fistula was successfully produced by the method described. A continuous murmur with systolic accentuation could be heard in the postoperative period in each case. The size of the fistula

which was produced in these animals varied from 4 to 10 mm. in diameter. The smaller fistulas (4 to 5 mm. in diameter) were well tolerated by the animals and there were no manifestations of cardiac failure during observation periods of one to four months in this group. Animals with aorticopulmonary fistulas measuring 6 to 10 mm. in diameter died with pulmonary edema in four to ten days after operation.

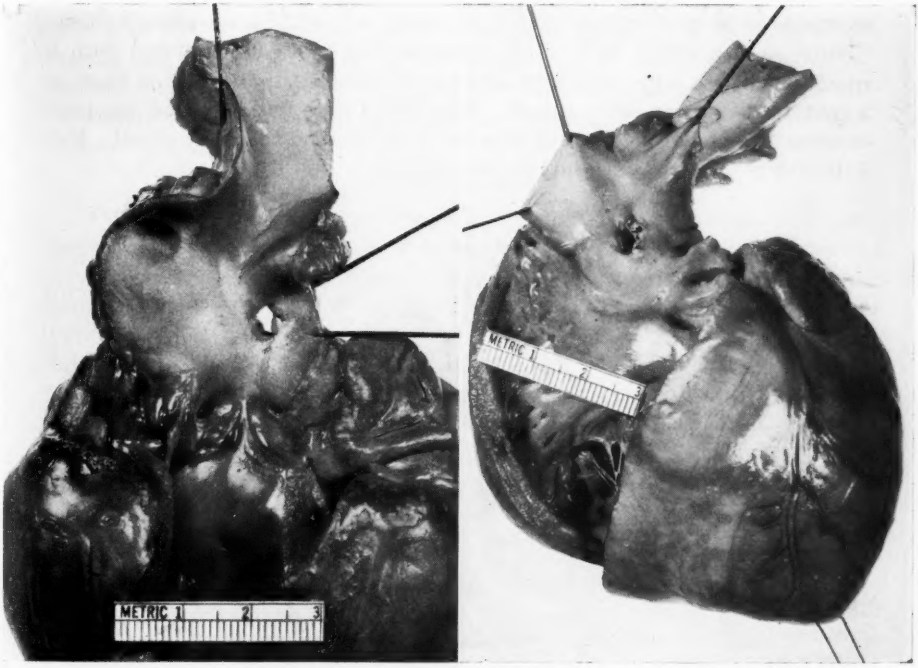

Fig. 3.—Photographs of dog's heart showing aortic and pulmonary aspects of small experimental fistula one month after its production.

Closure of Aorticopulmonary Fistulas.—In twelve of the dogs with experimental aorticopulmonary fistulas a second thoracotomy was done and an attempt was made to close the fistula. Because of the windowlike nature of aorticopulmonary fistulas in patients and the relatively large diameter of some of these congenital communications, it was felt that a technique of closure based on the use of encircling ligatures or suture ligatures would prove to be unsatisfactory in clinical application. Consequently, in these animals with experimental fistulas efforts were directed toward closure of the fistula by a method of division and suture. The procedure used was as follows. The animal was anesthetized with ether and the old thoracotomy wound was reopened. Adhesions between the lung and the pericardium were separated and the pericardium was opened, exposing the great vessels and the site of the fistula. Careful dissection was carried out to free the circumference of the aorta and

the main pulmonary artery, proximal and distal to the fistula. With the vessels mobilized as widely as possible the dissection was then carried around the fistula itself. A curved Potts ductus clamp was applied to the aortic side of the fistula grasping a crescentic segment of the aortic wall. In a similar manner a straight Potts ductus clamp was applied to the pulmonary artery so as to occlude the pulmonary aspect of the fistula. The clamps were applied in parallel to the long axis of the vessels. The fistula was divided between the clamps so as to leave a vascular cuff for suture within the grasp of each clamp. Closure of the defect in the wall of each vessel was accomplished with a running mattress suture of 00000 arterial silk which was returned on itself as a continuous over-and-over suture. The clamps were then removed, the pericardium was closed loosely, the lung re-expanded, and the chest closed. Fig. 4 illustrates the steps in closure by this method.

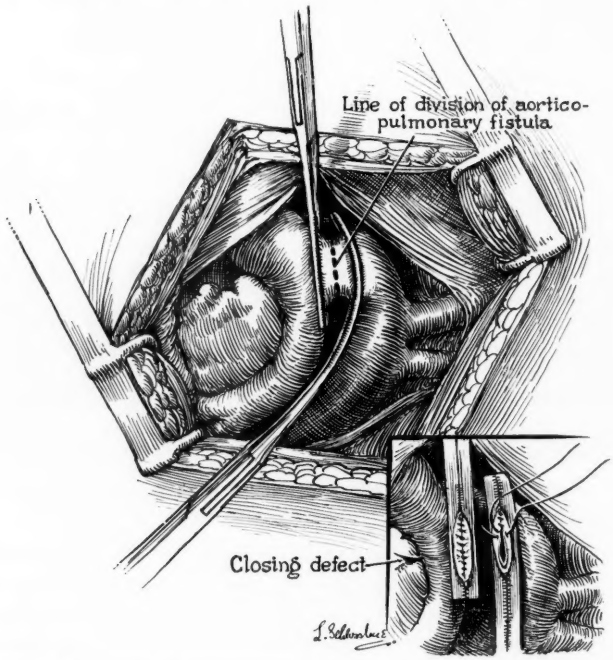

Fig. 4.—Technique of closure of experimental aorticopulmonary fistula by division and suture.

There are several possible variations in the method of application of the clamps and in the choice of straight and curved Potts forceps. However, the use of the curved forceps on the aortic side of the fistula permits a larger segment of aortic wall to be more easily grasped than when a straight clamp is used for this purpose.

Successful closure of the experimental aorticopulmonary fistula by this method was accomplished in five of the twelve dogs in which it was attempted.

The other animals were lost from hemorrhage at the time of attempted closure. Dense scar makes the dissection extremely difficult several months after the fistula is produced, and if closure is attempted within the first week or two after the anastomosis, friable granulations and easy dehiscence of the anastomosis add to the technical hazard. With a single exception, the successful closures were all accomplished one to three months after production of the fistula. These animals have been observed from one to six months since the closure of their experimental aorticopulmonary fistulas and have remained healthy and active. There have been no apparent ill effects from the slight degree of narrowing of the aorta and the pulmonary artery which has undoubtedly resulted from the production and closure of the fistula in the survivors.

CLINICAL ASPECTS

During an eighteen-month period (1950 to 1951) three patients with congenital aorticopulmonary fistula were studied and subsequently submitted to thoracotomy at the Johns Hopkins Hospital. The diagnostic problem presented by these patients and a detailed description of the clinical syndrome of aorticopulmonary fistula exceed the scope of this article and will be reported separately by Taussig and her associates.[10]

Cardinal clinical findings in the first two patients of this small series included absence of cyanosis and polycythemia, progressive cardiac enlargement, and a loud systolic murmur over the precordium, maximal in the third or fourth intercostal space to the left of the sternum. Cardiac catheterization indicated the presence of a large left to right shunt. A diagnosis of "atypical" ductus arteriosus was made in each case, although high interventricular septal defect and congenital aorticopulmonary fistula could not be excluded.

At operation in the first patient (female, aged 18 months) an aorticopulmonary fistula was found which measured 10 mm. in diameter (Fig. 1). The lower margin of the fistula lay 6 or 7 mm. above the pulmonary valves. During the dissection around the fistula, the base of the pulmonary artery was lacerated and the ensuing severe hemorrhage could not be controlled. At autopsy the fistula proved to be an isolated cardiac anomaly.

In the second patient (male, aged 4 years) thoracotomy revealed the malformation illustrated in Fig. 5. There was a right aortic arch and a large defect in the aortic septum which seemed to extend down to the level of the arterial valves. There was no sulcus at the base of the large common arterial trunk to indicate a point of demarcation between pulmonary artery and aorta. Direct measurements of pressure in the aorta and the pulmonary artery showed identical readings. No attempt was made to close this huge fistula.

Experience with these two patients stimulated our interest in devising a method of surgical treatment for congenital aorticopulmonary fistula and prompted the development of the experimental techniques which have been described. In the third patient the technique of closure of aorticopulmonary fistula by division and suture was applied successfully.

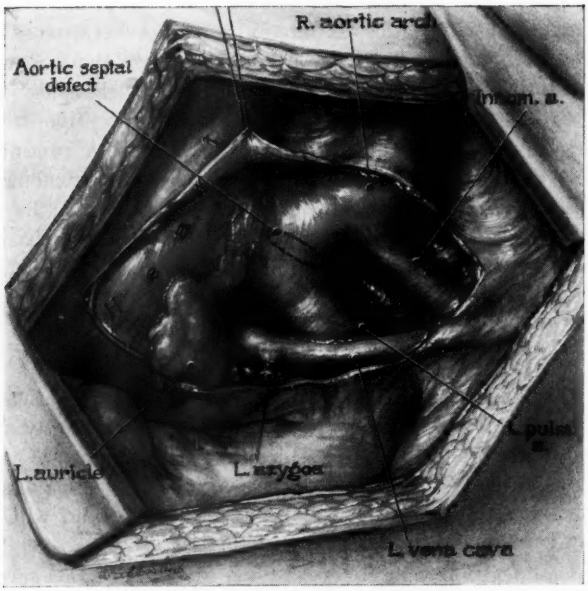

Fig. 5.—Sketch of findings at operation in 4-year-old boy with right aortic arch and large aorticopulmonary fistula which apparently extends to the level of the valves. Closure was not attempted.

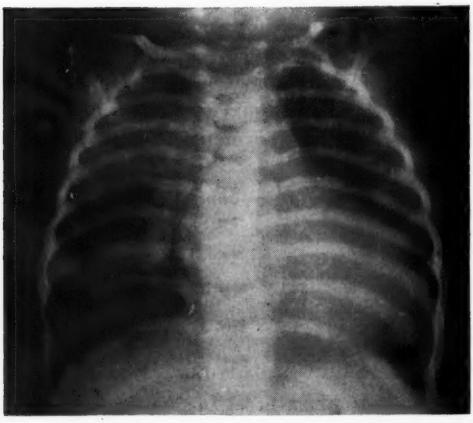

Fig. 6.

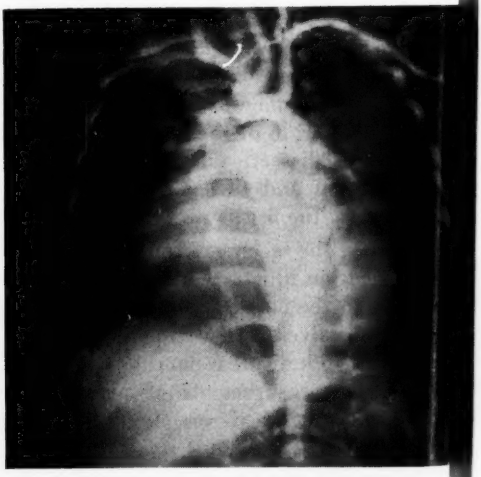

Fig. 7.

Fig. 6.—Reproduction of posteroanterior roentgenogram of chest before operation in 10-month-old infant with congenital aorticopulmonary fistula (patient D. S.)

Fig. 7.—Reproduction of preoperative retrograde thoracic aortogram in patient D. S., demonstrating aorticopulmonary fistula (left anterior oblique view).

CASE REPORT

D. S., a 10-month-old white female, was admitted to the Harriet Lane Home of The Johns Hopkins Hospital on Dec. 5, 1951, because of cardiac failure. The mother's pregnancy had been uneventful. Delivery occurred two weeks prior to term and the birth weight was 5 pounds, 9 ounces. Since the age of 2 months the infant had been followed in the clinic because of recurrent respiratory infection, dyspnea, tachycardia, and progressive cardiac enlargement. She had always been a feeding problem and failed to make a satisfactory gain in weight. Cyanosis had never been noted.

Physical Examination.—On admission examination revealed a thin, underdeveloped female infant with temperature 97°, pulse 130, respiration 40, and blood pressure 130/60. Head, eyes, ears, nose, and throat were normal. There were fine moist râles at both lung bases. The heart was enlarged to the right and left. The apical impulse was in the fifth left intercostal space in the anterior axillary line. There was a diffuse, grade 2 systolic murmur along the left sternal border, maximal in the fourth left intercostal space. The liver edge was palpable 3 cm. below the costal margin. There was no cyanosis, clubbing of the digits, or edema.

Fluoroscopy.—The heart was moderately enlarged (Fig. 6). The pulmonary conus was not prominent in appearance, but was rather more active than normal. The pulmonary hilar shadows were moderately increased and showed slight but definite pulsations. The peripheral lung fields were not remarkable. There was a left aortic arch.

Angiocardiogram.—Four cubic centimeters of 70 per cent Diodrast was injected into a vein in the right arm. Both anteroposterior and lateral exposures were made and films were taken at 1, 2, 3, 4, 5, 6, and 7 seconds. The contrast medium was seen to enter the right auricle through a normal superior vena cava. The right auricle was not enlarged. The right ventricle seemed to be of normal size and the pulmonary arteries received a normal amount of dye. The aorta was not visualized. No patent ductus was demonstrated.

Retrograde Thoracic Aortogram.—The left ulnar artery was cannulated and films were taken following the retrograde injection of 5 c.c. of 35 per cent Diodrast. On one film the entire thoracic aorta was well visualized (Fig. 7). The aorta was of normal size and contour. The contrast medium had regurgitated well down into the ascending aorta and there was definite opacification of the main trunk of the pulmonary artery and its branches, indicating the presence of an abnormal communication between the aorta and the pulmonary artery. No patent ductus could be identified.

Cardiac Catheterization.—Catheterization was attempted but because of technical difficulties the superior vena cava could not be cannulated. The inferior vena cava was entered via the saphenous vein but the development of a nodal rhythm necessitated the termination of the procedure.

Laboratory Studies.—Red blood count 4.71 million, hemoglobin 12 Gm. per cent, hematocrit 37.2. Electrocardiogram showed a rate of 100 with a sinus rhythm, P-R interval 0.18 seconds, QRS interval 0.02 second. The electrocardiogram was interpreted as showing a balanced axis with no definite ventricular hypertrophy and slight digitalis effect.

Course.—Despite digitalization the infant remained in cardiac failure with progressive enlargement of the heart and of the liver. After a review of all the data, Dr. Taussig and her associates made a diagnosis of congenital aorticopulmonary fistula and operation was advised.

Operation.—Operation was carried out by one of us (H. W. S., Jr.), Dec. 21, 1951.

Anesthesia.—Intratracheal oxygen-ether was used.*

Technique.—A cannula was inserted in an ankle vein for constant venoclysis and the chest was prepared and draped for bilateral anterior thoracotomy. A left submammary incision was placed, and the chest entered in the third intercostal space. Mediastinal dissection revealed no evidence of a patent ductus. There was a systolic thrill in the

*Administered by Miss Olive Berger.

pulmonary artery. The pericardium was opened widely and it was immediately apparent that there was a large fistula between the aorta and the pulmonary artery near the base of the heart (Fig. 8).

In order to obtain a more adequate exposure the cutaneous incision was extended across the sternum and over the third intercostal space on the right to a point just medial to the nipple. The sternum was divided with a Gigli saw, the internal mammary vessels were ligated and divided, and the entire wound was opened well into the right pleural cavity. A rectangular flap of pericardium was reflected to afford a wide exposure of the

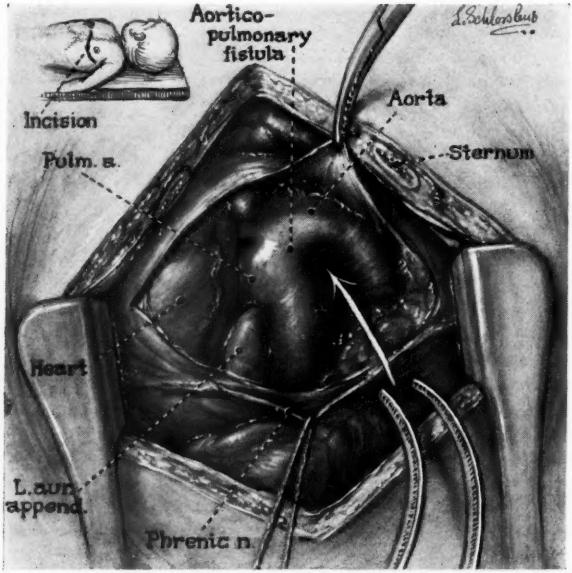

Fig. 8.—Sketch of anomaly as exposed at thoracotomy in patient D. S.

great vessels at the base of the heart. The aorta was normal in size but the pulmonary artery was larger than normal. There was a fistula between the aorta and the pulmonary artery which measured 16 mm. in its external diameter. The lowermost extent of the fistula lay 5 mm. above the base of the pulmonary artery and approximately 12 to 15 mm. from the base of the aorta. With the operator's finger in the transverse sinus, compression of the fistula bimanually readily delineated the elliptical margins of the communication between the two vessels and obliterated the thrill in the pulmonary artery. A careful dissection was then carried out to free the adventitia and pericardial reflexion from the aorta and pulmonary artery proximal and distal to the fistula. During this phase of the procedure there were two transitory episodes of asystole which responded promptly to the intraventricular injection of 2 c.c. of 10 per cent calcium chloride solution and cardiac massage. After the great vessels had been mobilized as widely as possible the dissection was carried in the plane anterior to the origin of the right pulmonary artery and posterior to the fistula so as completely to encircle the latter. A curved Potts ductus clamp was then passed into position on the aortic side of the fistula in such a way as to grasp a generous segment of the wall of the ascending aorta. A second curved Potts ductus clamp was placed in parallel with the first clamp and applied to the main pulmonary artery so as to occlude the pulmonary aspect of the fistula. Both clamps were closed to the third

ratchet and locks were applied. The fistula was divided between the clamps, and the defect in the aortic wall was closed with a continuous over-and-over suture, using 00000 silk on an atraumatic needle (Deknatel) in two rows. Upon removing the aortic clamp there was no bleeding. The very short cuff in the grasp of the clamp on the pulmonary artery was improved by applying a second curved clamp and removing the original one. The defect in the wall of the pulmonary artery was then closed with a continuous mattress suture of 00000 silk which was returned on itself as an over-and-over suture. There was no bleeding on removing the clamp (Fig. 9).

After the completion of these maneuvers the infant's heart action seemed to be improved and no thrill could be palpated in the aorta or the pulmonary artery. The pericardium was closed loosely. Catheters were placed in both pleural cavities and led out through stab wounds for water-seal drainage. The divided sternum was re-approximated with mattress sutures of chromic catgut, the lungs were re-expanded, and closure of the thoracotomy wound was carried out in layers.

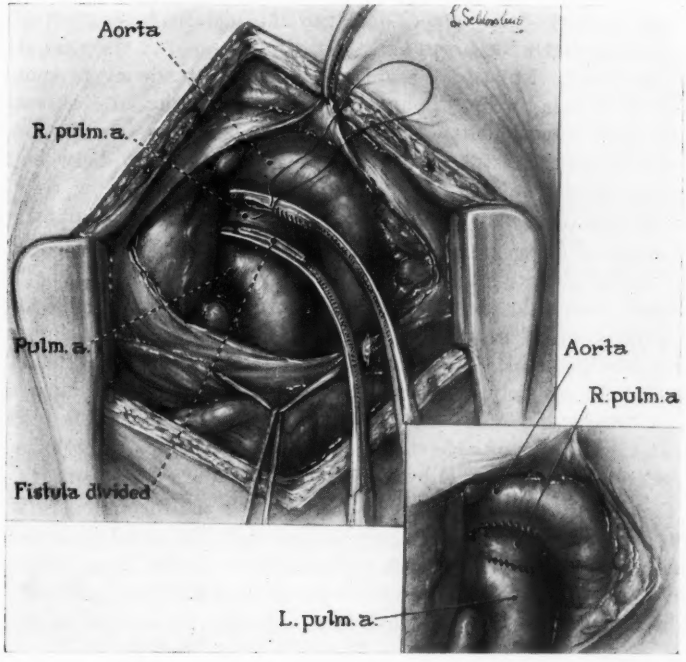

Fig. 9.—Closure of congenital aorticopulmonary fistula by disvision and suture using curved Potts ductus forceps (patient D. S.).

Postoperative Course.—The early postoperative course was characterized by moderate fever and tachycardia. The infant was maintained on penicillin, terramycin, and digitalis. After the first week the fever subsided, the infant became very active, took feedings well, and began to gain weight. With the disappearance of manifestations of cardiac failure digitalis was discontinued. The final roentgenogram of the chest prior to discharge showed some reduction in the size of the heart and clear lung fields. The patient was discharged from the hospital on Jan. 5, 1952. Since discharge she has been followed in the Cardiac Clinic by Dr. Taussig and her associates. In the five months since leaving the hospital the

baby has continued to do well with no manifestations of decompensation or other cardiac difficulties. A soft systolic murmur persists which is thought to be functional.

COMMENT

The experiments which have been described demonstrate the feasibility of constructing a suture anastomosis between the ascending aorta and the main pulmonary artery. The technique of producing an aorticopulmonary fistula might be suggested as an alternative to the Blalock and Potts anastomoses as a means of increasing the blood flow to the lungs in patients with tetralogy of Fallot. However, used in this capacity it would have no particular advantage over the other methods and would be a considerably more hazardous undertaking. The procedure has served to establish a satisfactory laboratory preparation which resembles congenital aorticopulmonary fistula in patients. In these experiments the animals exhibited a high degree of tolerance during the fifteen- to thirty-minute period of the anastomosis to degrees of partial occlusion of the ascending aorta and the main pulmonary artery ranging from 30 to 50 per cent. None of the animals showed a tendency to spontaneous closure of the fistula during the one- to four-month period of observation permitted by these experiments. Studies will be made to assess the long-range effects of this type of fistula and to investigate the relation between the size of the fistula and the volume of the left to right shunt.

The method of closure by division between Potts ductus clamps is simply an adaptation of Gross's original technique for division of patent ductus arteriosus[11] to the closure of an arterial fistula of the "window" type. In both the production and closure of the experimental fistulas and in the successful clinical application of the method of closure the Potts ductus clamps[12] have proved to be invaluable. The multiple fine teeth of these ingenious instruments will hold securely a segment of arterial wall without damage to the vessel. A narrow-bladed clamp of the Potts type, which offers a great advantage in the division of a short ductus arteriosus, is a necessity in the closure of an aorticopulmonary fistula by division and suture.

In the majority of the reported cases of this malformation in which an adequate description of the anomaly is included, the fistula has been quite similar anatomically to that which we have been able to close. The average diameter of the communication in most instances was in the range of 10 to 12 mm. The inferior margin of the fistula was usually several millimeters above the level of the pulmonary valve. Thus, it seems that the majority of congenital aorticopulmonary fistulas are amenable to surgical closure by the method which has been described. This method does not seem to be applicable without extreme risk to the closure of a huge fistula of the type which extends down to the level of the arterial valves as described by Dadds and Hoyle[13] or to the anomaly illustrated in Fig. 4. The presence of a sulcus, however small, which demarcates the lower extent of the fistula and permits dissection to be carried completely around the latter, is of considerable technical significance.

Surgical closure of congenital aorticopulmonary fistula in individual cases has been accomplished recently by Gross[14] and by Varco[15] by methods of liga-

tion or suture-ligation. The technique of division and suture would appear to offer a more accurate means of correction of a large windowlike communication between the great arterial trunks with less tendency to recurrence of the fistula. While undoubtedly rare as compared to patent ductus arteriosus, congenital aorticopulmonary fistula probably has a much greater incidence than indicated by the few reported cases. It is likely that the malformation will be recognized more frequently in the future with the current trend toward careful diagnostic evaluation of all forms of congenital heart disease.

SUMMARY

Congenital aorticopulmonary fistula is a rare cardiac malformation which results from an arrest in the development of the aortic septum. In an effort to devise a method of surgical treatment, the anomaly was reproduced in dogs. A technique for closure of the experimental fistulas by division and suture was worked out in the animals. This technique has been applied successfully in the closure of a 16 mm. aorticopulmonary fistula in a 10-month-old infant. Experimental and clinical aspects are briefly discussed.

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DISCUSSION

DR. AARON HIMMELSTEIN, New York.—Our group has been interested in the diagnosis of this lesion, particularly in differentiating it from the atypical patent ductus in which the physical findings are unusual. I would like to make the comment that in the catheterization studies, if one is able to thread a catheter through an aorta-pulmonary artery septal defect, it will go up the arch of the aorta and take the route that the arch

of the aorta takes, whereas if one threads a catheter successfully through a patent ductus it does not go through the arch of the aorta, but subtends that arch and goes down the descending aorta. This may be a feature that would help in the differential diagnosis of these two conditions.

DR. ROSS ROBERTSON, Vancouver.—I am intensely interested in Dr. Scott's excellent paper, because in operating on forty-five patients suspected of having patent ductus arteriosus I have encountered four similar cases. Regarding the name, I would like to take issue with Dr. Scott, possibly because I am a Canadian and favor Maud Abbott's terminology of aortic septal defect, which is a defect in the septum which divides the truncus arteriosus into the aorta and the main pulmonary artery.

I do not feel quite as sanguine about the preoperative diagnosis as Dr. Scott. I admire him for making the diagnosis in this case. Even postoperatively we have been unable to distinguish the cases definitely, in a clinical way, from a patent ductus, the reason being, of course, that they have the same physiologic effect as a patent ductus. We have come to feel that if you have a case of patent ductus which seems to be almost too good to be true, then you suspect this defect. If you have a child with symptoms of dyspnea, or undernourishment, or excess fatigue, and find an enlarged heart with a very loud continuous murmur placed a little more medially than that of a patent ductus; if you have a very high pulse pressure with the diastolic pressure frequently falling to zero; and on x-ray examination, in addition to the large heart you see that the pulmonary vascular markings are greatly accentuated—then, I think, you should suspect this defect.

When we encountered this lesion at operation, there was no patent ductus, but there was a thrill over the main pulmonary artery close to the heart. On opening the pericardium the thrill was found to originate about one inch from the heart; and the main pulmonary artery and aorta seemed to be fused together. Subsequently, suspecting this defect, we produced a similar defect on a dog and did aortograms which demonstrated the defect. Aortograms on one of these children showed the same type of defect as produced in the dog. We have not had the courage to proceed any further, suspecting that the defect continued down to the pulmonary valves. I am grateful to hear Dr. Scott describe the little notch found between the defect and the pulmonary valve and permitting the application of forceps for repair.

DR. ROBERT SHAW, Dallas.—About three years ago we saw a 5-year-old boy from a neighboring community who obviously had a congenital heart lesion. On fluoroscopy we learned that this child had enormous pulmonary arteries with a typical hilar dance. Angiocardiography was not very conclusive but our radiologist felt that the diagnosis was probably a large interatrial septal defect, although there were some features in the case that did not seem to be explained by this diagnosis. We advised the parents that as yet we had no surgical procedure we could offer the child. About a year and a half later this child suddenly died while playing in the back yard of his home, and at autopsy it was found that he had an aorticopulmonary fistula, and that there had been rupture of the pulmonary artery intrapericardially with sudden tamponade.

DR. FRANK GERBODE, San Francisco.—I wish to compliment Dr. Scott upon his approach to this very difficult problem. The experimental method he used in solving it is, I think, the one we should adopt more often in the solution of cardiovascular problems. We have encountered fistulous connections near the base of the aorta in five patients in whom exploration was carried out for patent ductus arteriosus. In each instance there was no ductus. One of these patients subsequently expired, and at necropsy we found a ruptured aneurysm of the sinus of Valsalva. (Slide.) This slide shows a small connection just above the valve where there was a fistula leading to the right ventricle. This produced a continuous murmur and blood pressure changes, like a ductus. Just beneath this there is a high septal defect which also contributed to the continuous murmur.

In our cases cardiac catheterization was not of great help, although it did show a higher oxygen saturation of the pulmonary arterial blood than is usual with a ductus. In

other words, the differences were greater than one might expect with a normal ductus. In general, the continuous murmur was louder than it is with a ductus. We were unable to close the opening in any of these patients because of the proximity of the connection to the coronary vessels or to the valves. I think this must be borne in mind in all these patients, because if one tries to close an opening low in the great vessels, one will encounter this difficulty. In Dr. Scott's case it was possible to establish the proximal limit of the opening. This case will serve to stimulate further efforts toward developing methods of closing these defects.

DR. SCOTT (Closing).—I would like to thank the discussants for their interesting remarks. Dr. Himmelstein's use of the catheter certainly should prove to be an excellent means of diagnosis if one is lucky enough to enter the fistula. I am very much impressed by the large incidence of this hitherto considered rare anomaly exhibited by the remarks of the discussants. I think there is one thing about the technical side of the attempts at closure of these defects that might be brought out; that is, that in the two cases we had an opportunity to examine, namely, the one at autopsy and the clinical case which was successfully divided, there was a difference in the proximity of the fistula to the aortic annulus and the annulus of the pulmonary valve. In other words, the base of the aorta lies lower than does the base of the pulmonary artery, so that the coronary arteries at their points of origin will be somewhat below the fistula if the fistula is associated with some kind of little sulcus to demarcate its lower extent. I believe this is an important technical point.

PULMONARY FUNCTION AFTER MULTIPLE SEGMENTAL RESECTION FOR BRONCHIECTASIS

RICHARD H. OVERHOLT, M.D.,* JAMES H. WALKER, M.D.** (BY INVITATION), AND BENJAMIN ETSTEN, M.D.*** (BY INVITATION)
BOSTON, MASS.

BRONCHIECTASIS may involve any of the eighteen segments which comprise the five lobes of the two lungs. The disease is frequently bilateral, but rarely is there involvement of all the constituent segments of any upper or lower lobe. The right upper lobe, the two top segments of the left upper lobe, and the superior segments of both lower lobes are seldom infected and usually compensate in size and function.

Severity of symptoms is not necessarily related to the extent of involvement, but relief of these symptoms is dependent upon the complete excision of the disease. The conservation of normal healthy tissue is as much a part of the fundamental concept of surgery as the eradication of the disease. The intent of this basic concept is achieved by segmental resection in surgery for bronchiectasis. Segmentectomy presents the problem of greater technical difficulty and more diligent postoperative attention, but the ultimate advantage of preserving healthy pulmonary tissue makes it desirable for the unilateral case as well as the severe bilateral case.

The purpose of this paper is to present the results of postoperative pulmonary function studies on patients with segmental resection for bilateral and unilateral bronchiectasis.

METHOD OF STUDY

The results of this investigation comprise data from a group of fourteen unselected patients who had either unilateral or bilateral segmental resection one to seven years prior to study. None of these patients had preoperative pulmonary function studies. All of them were admitted to the hospital for study. Pulmonary physiologic determinations were performed in the fasting state. Total spirometry was done in the morning and differential bronchospirometry in the afternoon. Arterial bloods were drawn for gas analyses and pH determinations during the resting state. On another day, total lung volume measurements were obtained.

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Ventilation Studies.—Total vital capacity was performed with modified Benedict-Roth spirometer, and the timed capacity was measured with a vital capacity spirometer.[1] Oxygen consumption, tidal volume, respiratory minute volume (R.M.V.), inspiratory capacity, and expiratory reserves were obtained using a balanced spirometer with soda lime in the circuit. The maximum breathing capacity (M.B.C.) was obtained using a Hans-Rudolph high velocity respiratory valve and a Douglas bag.[2] The gas was collected for thirty seconds, measured by a Tissot spirometer, and reported in liters per minute at 37° C. wet. The best of three efforts is reported. The walking ventilation test consisted of a level walk at the rate of 180 feet per minute for three minutes.[3] The expired air was collected in a Douglas bag; the volume was measured like that of the M.B.C. and recorded in liters per minute at 37° C. wet. The walking index was calculated from the equation: $\frac{\text{walking ventilation}}{\text{M.B.C.}} \times 100$.[3] The predicted M.B.C. and vital capacity (V.C.) were calculated according to Cournand's regression formulas.[4] The air velocity index was calculated from the equation: $\frac{\text{per cent of predicted M.B.C.}}{\text{per cent of predicted V.C.}}$.[5] The ventilation equivalent for oxygen was calculated from the equation: $\frac{\text{minute ventilation in liters}}{\text{minute oxygen uptake in c.c.}}$ This is reported as the number of liters ventilated per 100 c.c. oxygen removed. The breathing reserve may be obtained from the equation: $\frac{(\text{M.B.C.}-\text{M.V.})\ 100.}{\text{M.B.C.}}$.[6]

Differential Bronchospirometry.—The patients were sedated with 75 mg. pentobarbital sodium and 0.6 mg. scopolamine thirty minutes before the start of the procedure. Laryngo-tracheo-bronchial anesthesia was obtained with 4 per cent cocaine. A Carlens double-lumen catheter was inserted under fluoroscopic guidance.[10] Records of oxygen consumption, tidal exchange, respiratory minute volume, inspiratory reserve, expiratory capacity, and vital capacity were obtained on a twin recording spirometer.

Arterial Blood Studies.—Arterial oxygen content, capacity, and carbon dioxide determinations were made on 1 c.c. samples of whole blood of the method of Van Slyke and Neill.[7] The arterial blood for pH was determined by a Beckmann model G glass electrode. The pH was corrected for a temperature of 38° C.[8] The carbon dioxide tension was calculated from the carbon dioxide content in volume per cent and the pH value from the nomogram of Peters and Van Slyke.[9] Total carbon dioxide values in millimeters per liter were read from the chart of Peters and Van Slyke.[10]

Total Lung Volume.—The patient was allowed to rest in the supine position for thirty minutes previous to the measurement of function residual capacity (F.R.C.). The Darling[11] open circuit with slight modification was used. After the first determination, the inspiratory capacity, residual capacity, and vital capacity were recorded on a Warren Collins respirometer. After a two-hour interval of rest, these tests were repeated. The following calculations were

made from these determinations: functional residual capacity, residual volume, total capacity, and alveolar nitrogen content after seven minutes of oxygen breathing.

CASE HISTORIES

These four cases serve to illustrate the value of segmentectomy in bronchiectasis by eradication of disease and conservation of healthy pulmonary tissue.

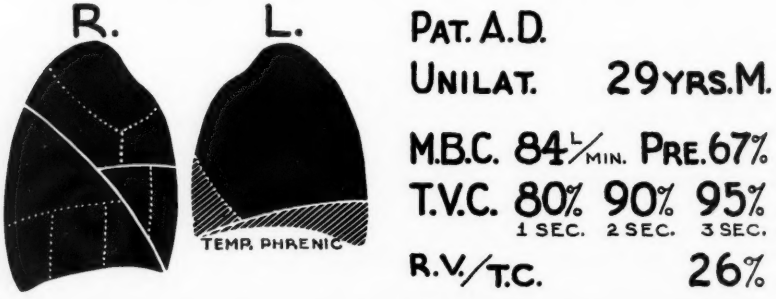
CASE 1.—Patient A. D. had unilateral bronchiectasis with a chronic abscess since the age of 3 years, which involved the left lower lobe, the lingula, and anterior segments of the left upper lobe. The involved segments were excised in January, 1952.

The studies in this patient are of fundamental significance because they provide the opportunity to evaluate the function of an individual bronchopulmonary segment.

The maximum respiratory muscular effort (M.B.C.) of 84.4 L. per minute, 67 per cent predicted, indicates that the total function is efficient for the total number of remaining segments.

This patient had a total lung capacity of 3.8 L., and the residual volume was 26 per cent of his total capacity. This indicates that emphysema is not present. The alveolar nitrogen index of intrapulmonary mixing is normal, further evidence of normally functioning lung tissue without emphysema.

The functional relationship between the one segment on the left and the ten segments of the right lung show that the one segment contributes 22.6 per cent of the total oxygen consumption and 28 per cent of the ventilation. This clearly demonstrates the value of conserving one segment which could be lifesaving in any respiratory emergency.



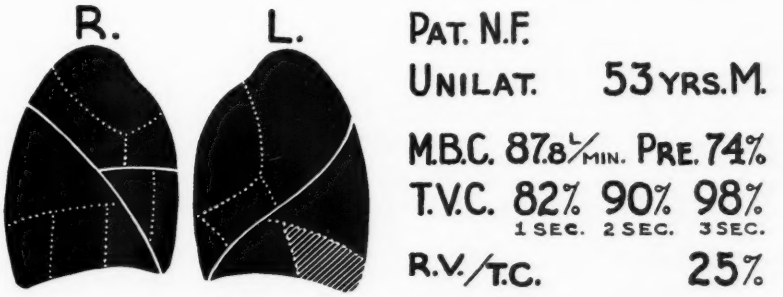
	RIGHT 10 SEG.	LEFT 1 SEG.
O_2 Cons. %	77.4	22.6
Vent. %	72	28
V.C. %	80	20
Vent. Eq. L./min.	2.4	3.2

Fig. 1.

CASE 2.—Patient N. F. had bronchiectasis involving the posterior and lateral basal segments of the left lower lobe. An empyema at the left base had been drained in 1930. Following this, he developed a cough and occasionally some purulent sputum, and on three occasions he had minimal drainage through the previous operative site. The involved segments were removed, and postoperatively there was a moderate air leak from the segmental surfaces for forty-eight hours.

The pertinent problem in this case was either to resect the entire left lower lobe or conserve the normal segments and remove only the diseased tissue. The removal of the two abnormal segments allowed the remaining segments of that lobe to participate with the left

upper lobe in 36.6 per cent of the total oxygen consumption. The left hemidiaphragm was elevated and fixed due to surgery for empyema in 1930, and this may explain the decrease in vital capacity and the elevated ventilation equivalent as compared to the right lung. Residual volume and intrapulmonary mixing volume were normal. The removal of two additional normal segments most likely would have further impaired the function of the left lung.



	RIGHT 10 SEG.	LEFT 6 SEG.
O_2 Cons. %	63.4	36.6
Vent. %	59.1	40.9
V.C. %	68.8	31.2
Vent. Eq. L./min.	4.64	5.56

Fig. 2.

CASE 3.—Patient G. Z. was markedly incapacitated from severe bilateral basal bronchiectasis since early childhood. The left basal segments were excised in February, 1950, and the right basal segments in June, 1950. There were no postoperative complications.

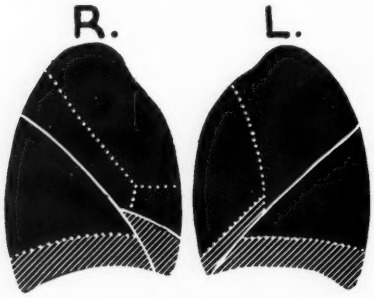
The resection of only the diseased segments has not impaired pulmonary function. This is well demonstrated by total function studies, differential bronchospirometry, and arterial blood gases.

	RIGHT 6 SEG.	LEFT 5 SEG.
O_2 Cons. %	47	53
Vent. %	54	46
V.C. %	55	45
Vent. Eq. L./min.	5.47	4.1

Fig. 3.

CASE 4.—Patient H. K. had the typical bilateral bronchiectatic pattern—basal segments bilaterally, right middle lobe, and lingula. In addition to severe cough, excessive amounts of purulent sputum, and dyspnea, she had severe recurrent pulmonary hemorrhages. These symptoms developed at the age of 14 or 15 years. The abnormal segments on the right were removed in October, 1948, and those on the left in April, 1949. The postoperative course was normal.

The M.B.C. of 54 L. per minute on this patient with only seven pulmonary segments may be considered as evidence of good ventilatory function. The timed vital capacity reveals normal speed of air movement. Although the residual volume is 29 per cent of total capacity, the intrapulmonary mixing index is within normal range. This indicates that the patient has some overdistention or compensatory emphysema without impairment of gas distribution. The differential bronchospirometry indicates essential normal distribution of oxygen consumption, ventilation, and V.C. among the remaining segments. The left lung, however, ventilates more efficiently than the right.



	RIGHT 4 SEG.	LEFT 3 SEG.
O_2 Cons. %	58.2	41.8
Vent. %	67.3	32.7
V.C. %	60	40
Vent. Eq. L./min.	3.1	2.1

Fig. 4.

RESULTS

The total results of this study are summarized in Tables I, II, and III. In the group with unilateral resection, the total function studies show that the fewer the number of segments excised the greater the percentage predicted of M.B.C. The walking ventilation, W.V./M.B.C. ratio, ventilation equivalent, and the speed of air flow on expiration in all the cases of this series were within normal limits.

A comparison of the function of the normal lung and that of the operative side (Table III) illustrates the following:

1. The percentage of oxygen consumption and ventilation increases with the number of segments conserved.

2. Ventilation was slightly increased over that of oxygen consumption on the operative side. This slight increase may be considered as a factor of the method, rather than impairment of segmental function.

In the group with bilateral surgery, the parallel increase of percentage M.B.C. with the increase of number of remaining segments is similar to that of

TABLE I. VENTILATION—POSTSEGMENTAL RESECTION

PATIENT	AGE	SURGERY	NO. REMAINING SEGMENTS	INTERVAL		M.B.C.		TIMED V.C. (%)			WALKING VENT.	W.V. M.B.C.	A.V.I.	REMARKS
				YR.	MO.	L./MIN.	PRED. %	1 SEC.	2 SEC.	3 SEC.	(L./MIN.)			
						Unilateral								
F. M.	40	L basal, lingula	13	2	3	56	69	80	88	94	11.9	0.17	0.84	Has bilateral bronchiectasis
M. M.	23	RML	16	2	5	145	92	90	95	98	21.0	0.15	0.93	Obliterated fissures, segmental technique used
E. C.	30	LLL lingula	12	0	9	68	98	75	94	100	17.1	0.25	0.98	
L. R.	47	Lingula, sup. seg. LLL	15	1	6	66	88	78	92	96	13.9	0.21	0.98	
N. F.	53	Post. & lat., basal LLL	16	0	7	88	74	82	90	98	22.5	0.25	0.93	
A. D.	29	LLL lingula & ant. LUL	11	0	2½	84	67	80	90	95	18	0.21	0.90	Left temporary phrenic
						Bilateral								
G. Z.	29	L basal, R basal	11	1	7	104	83	76	94	98	16.9	0.16	1.10	
A. R.	27	RML, RLL, L basal	8	0	7	80	140	70	85	90	25.0	0.31	1.0	
F. O.	43	LLL, lingula, RML & rt. basal	6	1	6	67	55	--	--	--	13.8	0.21	0.59	
H. K.	29	L basal & lingula, R basal & RML	7	1	11	55	58	78	85	100	21.5	0.39	0.84	
R. H.	28	LLL, lingula, RML	10	2	0	75.2	91	80	85	95	19.7	0.26	1.79	
C. F.	34	LLL, lingula, RML & ant. RUL	9	3	1	73	81	--	--	--	12.8	0.17	1.16	
H. M.	33	RML, lateral basal RLL, L basal, inf. lingula	11	3	2	56.8	78	76	91	95	17.0	0.30	0.94	
R. D.	39	LLL, lingula, R basal, inf. subseg. of ant. seg. of RUL, RML	5½	-	-	39	53	90	95	100	12.2	0.31	1.2	

TABLE II. LUNG VOLUMES—POSTSEGMENTAL RESECTION

PATIENT	REST RESP. RATE /MIN.	TIDAL VOL. (C.C.)	MIN. VOL. (L./MIN.)	O_2 UPTAKE (C.C./MIN.)	VENT. EQUIV. (L./MIN.)	VITAL CAP. (C.C.)	INSP. CAP. (C.C.)	EXP. RES. (C.C.)	FUNCT. RESID. CAP. (C.C.)	RESID. CAP. (C.C.)	T. LUNG CAP. (C.C.)	RV/TC %	MIXING INDEX $\%N_2$
Unilateral													
F. M.	7	746	5.2	208	2.6	2274	1156	888	----	----	----	--	- --
M. M.	12	723	8.3	448	1.86	4438	2932	1527	1645	118	4556	3	0.77
E. C.	22	376	8.4	325	3.2	1799	1657	326	917	591	2390	25	0.55
L. R.	11	806	8.8	314	2.8	2120	1640	393	----	----	----	--	- --
N. F.	20	668	13.6	453	3.2	3076	2307	789	1843	1054	4130	25	1.13
A. D.	14	748	10.4	369	2.8	2932	2265	689	1914	1044	3976	26	1.20
Bilateral													
G. Z.	12	607	7.6	326	2.4	3107	1893	1174	2215	1041	4148	25	0.67
A. R.	20	397	7.9	334	2.4	2883	1722	1019	2759	1740	4623	38	2.46
F. O.	--	630	6.3	270	2.3	3760	2290	1060	----	2470	5900	42	- --
H. K.	12	728	8.7	304	2.5	2023	1376	749	1584	835	2858	29	1.03
R. H.	22	346	7.6	256	3.0	1496	916	631	1150	519	2015	26	1.37
C. F.	--	445	5.3	179	3.1	2139	1116	883	----	----	----	--	- --
H. M.	17	446	7.5	248	4.4	2393	1521	1014	1763	749	3142	24	1.15
R. D.	15	348	5.2	241	1.9	1267	879	552	1322	666	1933	35	1.11

TABLE III. DIFFERENTIAL BRONCHOSPIROMETRY AND ARTERIAL BLOOD GASES—POSTSEGMENTAL RESECTION

		RIGHT LUNG					LEFT LUNG					RESTING, ARTERIAL BLOOD GASES			
PATIENT	TOTAL SEG.	SEG.	$O_2\%$	VENT. %	V.C. %	V. EQ. (L./MIN.)	SEG.	$O_2\%$	VENT. %	V.C. %	V. EQ. (L./MIN.)	O_2 SAT. %	pH_s 37° C.	pCO_2 (MM. HG.)	TOTAL CO_2 (MM./L.)
Unilateral															
F. M.	13	10	59.6	58.5	63.9	4.2	3	40.4	41.5	36.1	4.3	-- -	- --	-- -	-- -
M. M.	16	8	45.8	52.7	49.9	2.2	8	54.2	47.3	50.1	1.7	95	7.53	33	27
E. C.	12	10	81.2	73.8	77.3	2.1	2	18.8	26.2	32.7	3.2	94.6	7.45	33.5	23.2
L. R.	15	10	70	58.1	66.2	2.16	5	30	41.9	33.8	3.65	97	7.25	48.8	21.3
N. F.	16	10	63.4	59.1	68.8	4.64	6	36.6	40.9	31.2	5.56	94	7.38	49	23.4
A. D.	11	10	77.4	72	80	2.4	1	22.6	28	20	3.2	95	7.45	39	26.7
Bilateral															
G. Z.	11	6	47	54	55	5.47	5	53	46	45	4.1	96	7.41	40.5	25.4
A. R.	8	3	39.3	38	45.7	3.9	5	60.7	62	54.3	1.5	97	7.41	30	19.5
F. O.	6	4	59.5	66.4	62.9	5.6	2	40.5	33.6	37.1	4.2	-- -	- --	-- -	-- -
H. K.	7	4	58.2	67.3	60	3.1	3	41.8	32.7	40	2.1	95	7.53	33	27
R. H.	10	8	66.7	71	72	4.06	2	33.3	29	28	3.03	96	7.36	45.5	25.5
C. F.	9	7	76.5	47.6	62	1.9	2	23.5	52.4	38	5.8	-- -	- --	-- -	-- -
H. M.	11	7	58	48	51	2.8	4	42	52	49	4.2	92	7.55	29	25
R. D.	5½	3½	-- -	-- -	-- -	- --	2	-- -	-- -	-- -	- --	-- -	- --	-- -	-- -

the unilateral group. Mild clinical dyspnea was evident in only one case (A.R.) following the exercise test. This patient was the only one of the group who had a timed vital capacity of 70 per cent in one second (normal value 75 per cent). The oxygen consumption is greater in the lung that has the greater number of segments (Table III). In five of the seven cases, on the side with the least number of remaining segments, the ventilation is less than the percentage of oxygen consumption.

Arterial blood gases were obtained in ten of the fourteen patients. The arterial oxygen saturation, pH_5, pCO_2, and total CO_2 were within normal limits on all of the patients studied (Table III).

Total lung volumes were obtained on eleven of the fourteen patients and the results are listed in Table II. In the unilateral surgical group all of the values were within normal limits. In the group with bilateral surgery, four patients had an increase in residual volume. Only one patient of the entire group had an elevated alveolar nitrogen value after seven minutes of oxygen breathing.

DISCUSSION

Clinical.—Prior to surgery, none of the six patients with unilateral surgery were incapacitated so that they could not perform their normal, everyday activities. Following surgery, they did not notice any remarkable change in their clinical breathing reserve or exercise tolerance, but did note a disappearance of symptoms.

Patient F. M. in the unilateral surgery group did have bilateral bronchiectasis but only the diseased segments on the left (basal segments and lingula) were resected. The symptomatic improvement was so marked that surgery on the right was deferred. The differential study indicates that the three segments on the left side now contribute approximately 40 per cent to the total oxygen consumption and ventilation as compared to the ten segments on the right. Of the ten segments in the right lung, six of these are bronchiectatic and presumably are not contributing much to the patient's pulmonary function.

Patient L. R. had frequent, severe pulmonary hemorrhages, and bronchography demonstrated cystic bronchiectasis of the lingula and the superior segment of the left lower lobe. If the unit of excision had been lobe instead of segment, it would have been necessary to excise the entire left lung to do away with this patient's symptoms. She now has fifteen segments remaining in the two lungs, with ten on the right and five on the left, and a differential study indicates that the remaining five segments on the left contribute about one-third of the total function.

Of the eight patients with bilateral bronchiectasis, only two were able to carry on any resemblance of normal activity prior to surgery. Clinically, they were short of breath, complained of cough, excessive, purulent sputum, and, in several cases, severe pulmonary hemorrhages. They have noted marked improvement in their exercise tolerance and breathing reserve, and also a disappearance of the symptoms since surgery.

Patient A. R. had extensive saccular bronchiectasis involving the right middle lobe, right lower lobe, and basal segments of the left lower lobe. He had been unable to attend school or to work because of the severe symptoms. Since surgery, this patient now works an eight-hour day, climbs two flights of stairs without dyspnea, and is free of cough and sputum.

Patient F. O. is a 43-year-old priest who now has only six segments, or one-third of the total number in normal individuals. Four of these are on the right and two on the left and postoperative bronchograms are normal. Differential studies showed normal distribution of oxygen consumption, ventilation, and vital capacity. His M.B.C. is 66.9 liters per minute, ventilatory equivalent is 2.36 liters, and he does have a residual volume of 42 per cent of his total capacity, indicating some pulmonary emphysema or overdistention. This degree of emphysema has not caused clinical dyspnea, and this patient can perform his required activities now for the first time in many years.

Patient C. F. had extensive bronchiectasis involving the left lower lobe, lingula, right middle lobe, and the anterior segment of the right upper lobe. The left lower lobe was removed in December, 1943, the lingula in 1947, and the involved segments on the right in 1948. She now has only 50 per cent of her total number.

Pulmonary function studies were first done in 1947 (prior to lingulectomy) and showed a M.B.C. of 74 L. per minute or 80 per cent predicted, walking ventilation of 12.5 L. per minute, and ventilation equivalent of 2.9 L. per minute. These studies were repeated following a lingulectomy, right middle lobectomy, and resection of anterior segment of the right upper lobe. It can be seen that removal of the additional segments (three on right, two on left) did not materially impair the mechanism of ventilation as shown by the M.B.C. The functional relationships between the right and left lung were also not altered. This case clearly demonstrates the beneficial effects of segmental surgery; namely, (1) eradication of diseased segments plus symptoms, and (2) conservation of normal pulmonary tissue and function.

Physiologic.—Preoperative pulmonary function studies were not obtained in this series for the following reasons:

1. The presence of abnormal amounts of secretions in the bronchial system obstructs air flow in bronchi of normal segments and causes an increase in ventilatory effort.

2. Excessive secretion impedes oxygen consumption.

3. In unilateral cases, the normal lung compensates for the altered function of the involved side.

Postoperative pulmonary function studies in the unilateral and bilateral groups indicate that segmental pulmonary resection does conserve the integrity of a large number of factors related to ventilation. A comparison of the determined M.B.C. to predicted value is recorded in Fig. 5. As there is an increase in the number of healthy segments, the determined M.B.C. approaches the predicted value for normal. This only emphasizes the im-

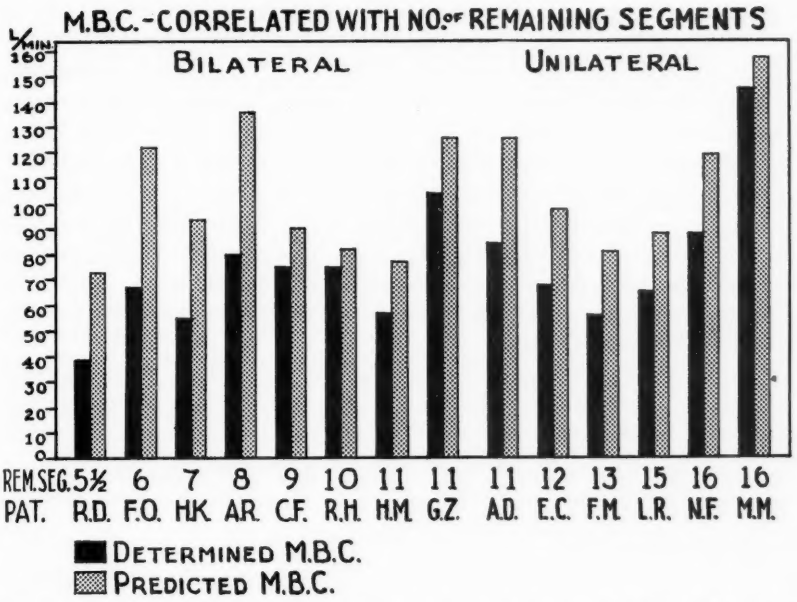


Fig. 5.—The bilateral group includes Patients R. D. to G. Z.; the unilateral, Patients A. D to M. M.

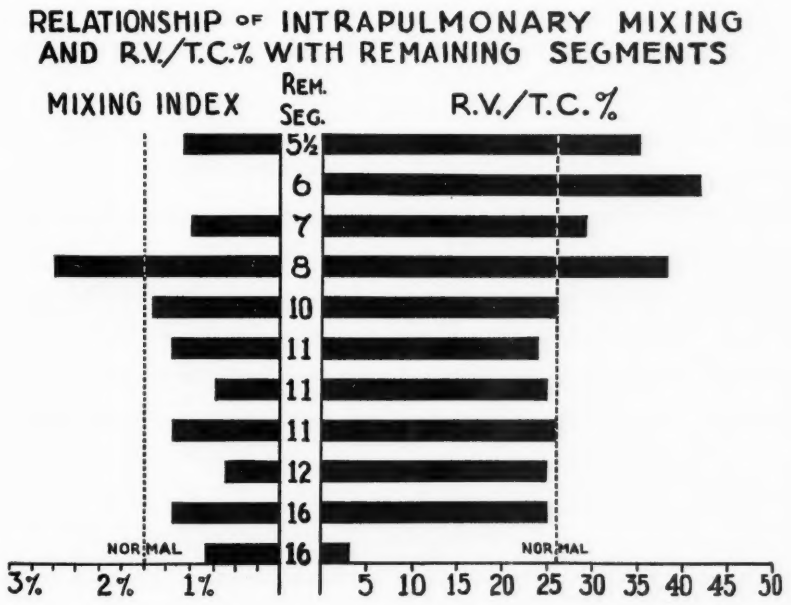


Fig. 6.

portance of the segments as the unit of excision in treating bronchiectasis. The determined M.B.C. is low in some of the patients but these values do not correlate with their clinical respiratory reserve. This supports Comroe's[12] contention that reduction in M.B.C. must be large to be significant because there can be individual deviation of 25 to 35 per cent of mean group values. Each conserved segment contributes to oxygen consumption and ventilation. This is well demonstrated in patient A.D. The one remaining segment on the left side participates in 21 per cent of the total oxygen consumption. The comparison of percentage oxygen consumption to percentage ventilation of the conserved pulmonary segments demonstrates efficient function. In the bilateral group, the side that had the greater number of segments removed ventilated within normal limits.

The four patients with an increased residual volume had the least number of remaining segments—5½, 6, 7, and 8 (Fig. 6). These findings are consistent with overdistention and most likely represent hyperinflation rather than true emphysema. The normal alveolar nitrogen values represent adequate intrapulmonary mixing of gases (Fig. 6). This supports the belief that hyperinflation does not cause pulmonary disability.

Dissection of the diseased pulmonary segments with a minimum of operative manipulation of the healthy segments, plus a minimum of postoperative respiratory complications, are important etiological factors in preserving normal function in the remaining lung tissue.

CONCLUSION

The pulmonary function studies in this series, following unilateral and bilateral segmental resection for bronchiectasis, demonstrate the necessity of saving normal lung segments.

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DISCUSSION

DR. J. GORDON SCANNELL, Boston.—I should like to confine my remarks to one aspect of Dr. Overholt's excellent presentation. It has occurred to us that one may question the propriety of a simultaneous bilateral resection on other than technical grounds. It has been both my training and my experience that in bronchiectasis one should be guided more by the demonstrated liability of a segment to a patient rather than by the radiologic diagnosis. We have frequently found that in a proposed bilateral program we have done one side and found it unnecessary to do the second side. Particularly has this been true in the scattered, radiologically involved segments which, in fact, may be merely scars of disease and may actually be doing some good in a patient's chest by occupying space.

DR. C. E. O'BRIEN, Ann Arbor.—The matter of bilateral pulmonary resection has raised certain problems which have been of considerable interest to us. There is, first, the question of how much pulmonary tissue may be removed allowing the operation to be compatible with life and with a reasonable degree of activity. Second, there is the matter of what happens to the remaining pulmonary tissue, and one thinks of the problem of overdistention as well as the pulmonary vascular resistance which may develop, with its effect on the function of the right heart. Third, we have been interested in the matter of the optimum time for operation upon the second side, and I am much interested to hear the idea proposed of simultaneous operations.

Our series is a long-range study, of course, but thus far our cases have demonstrated certain findings rather consistently. First, each patient who has had bilateral resection has demonstrated some degree of impairment of ventilatory function, diminution of vital capacity, and maximum breathing capacity. Second, each patient has shown some overdistention of the remaining pulmonary tissue as evidenced by the increase in the ratio of residual air to the total capacity. Third, there has been a tendency toward a low resting arterial oxygen saturation which is accentuated by exercise. And last, there have been changes in the ventilation equivalent suggesting that the function of the remaining pulmonary tissue is somewhat less efficient than normal.

I have a few slides which I thought might illustrate one or two things we have observed: (Slide.) This is a young woman 20 years of age who had thirteen segments removed, having had the lingula of the left upper and the left lower and also the right middle and right lower lobes removed. You will notice that there is a diminution in ventilatory function and there is some increase in residual air. I think probably the latter is ominous from the point of view of what is going to happen to this young woman in years to come. You will notice that while there is slight evidence of arterial oxygen desaturation, it is not striking. This young woman lives a normal life; she works full time and enjoys dancing and bowling and such activities.

(Slide.) On the other hand, this patient is 37 years of age, with both lower lobes removed. This illustrates, we believe, certain important principles. The man was recognized to have, in addition to bilateral lower lobe disease, a minimal amount of bronchiectasis in the anterior segment of the left upper lobe. It was thought to be minimal, but when that segment of the left upper lobe lost the advantage of its position so far as adequate drainage is concerned, he began to have much more severe symptoms from that bronchiectasis which was left behind. You will note that there is a relatively severe degree

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of impairment, particularly in the maximum breathing capacity, less in the vital capacity; the intrapulmonary mixing is very poor, and you will notice the great degree to which this man desaturates the arterial blood on exercise. We wonder about the point at which the function is lost in patients such as this. I might say that this man suffered a complicated postoperative period with bronchial fistula on the left side which prolonged the convalescence and resulted in certain pleural changes, which we think are responsible for some of this impairment of function, as is the residual disease.

(Slide.) So to get at the basis of this we thought that it would be interesting to know how the remaining pulmonary tissue on the side of resection functions immediately after operation. These are observations which were made immediately after operation with the patient still under anesthesia. We were surprised to notice the relatively good function of the remaining pulmonary tissue. In some cases the oxygen uptake was actually increased, in other cases in which abnormal tissue was removed the ventilation equivalent was improved, and in general it may be said that the immediate postoperative function, so far as can be determined, is surprisingly good. We think this emphasizes the point which has been mentioned by others, namely, that what happens to the pleura because of complications in the postoperative period is of exceedingly great importance in the final functional picture.

In summary, I would like to say that I am in complete agreement with the principle of preserving all normal pulmonary tissue. I think this is important from the point of over-all function obtained from the segments which are preserved, and it is also important from the point of view of taking up the space which would have to be filled by overdistention of the remaining segments, if normal segments were removed simply for the purpose of doing a total lobectomy rather than segmental resection. Second, one must beware of bronchiectasis which appears to be of little significance initially, which is in a favorable position for bronchial drainage from the upper lobes, but which loses this favorable position and becomes dependent and symptom-producing when it serves to fill the space it must occupy after lower lobe resection. Third, I will reiterate that early postoperative pulmonary function is surprisingly good in the segments remaining on the side of operation, and one must bend every effort to prevent postoperative pleural complications in an effort to preserve this function.

DR. OSLER ABBOTT, Atlanta.—I should like to mention one factor in regard to resection and the loss of a major amount of lung tissue in the presence of a large amount of dead space. It is a very simple matter to make a suture purse string in the diaphragm and insert a catheter into the peritoneal cavity, creating a pneumoperitoneum just before closing the chest. His comfort, postoperatively, is increased; his diaphragmatic function and his temporary pulmonary disturbance from overdistention of the lung will be greatly aided.

DR. OVERHOLT (Closing.).—Dr. Scannell has brought up a very important point about the evaluation for the need of doing the second side in bronchiectasis. It happens that we have seen some 220 patients with bilateral bronchiectasis and elected to do bilateral surgery in only seventy cases. I agree that it should be clearly evident that bilateral resection will be required before such treatment is planned. Clinical evaluation, character of the bronchial dilatations, and bronchoscopic study may clearly indicate that the extent of the septic reservoir on both sides requires removal. It is then possible to tell in the beginning that a particular patient may or may not need to have both those septic reservoirs removed. In the selection of cases or in proposing surgical treatment, every surgeon must place himself in the position of the patient and do for him what he thinks he would like to have done for himself. In the particular cases we selected for bilateral resection, it was decided that if I were in the patient's place, I would like to have my problem managed in that particular way. I should point out, however, that probably the number of cases that may be handled that way are very few, and each surgeon has to make his own decision and work out his own solution for the problem that will provide

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the greatest factor of safety. We did it in order to eliminate the spillage from the contralateral side.

It might have been interesting if we could have had preoperative function studies made in all of the cases included in this report. There were four reasons why we did not have these studies: first, the presence of abnormal quantities of secretion in the bronchial system obstruct air flow in normal segments and cause an increase in ventilatory effort; second, excessive secretion impedes oxygen consumption; third, in the unilateral cases, the normal lung compensates for altered function of the involved side; fourth, at the time most of these studies were done, we did not have the physiology laboratory setup to do the studies—which fact alone provides an adequate reason for not having had preoperative studies in these particular patients.

Dr. O'Brien, in his very excellent discussion, brought out an interesting point. That is, that in the very early studies of function, immediately after partial lung resection, function as measured may be extremely good and that possibly some of the late disturbances in function are most likely due to the effect of pleural constriction. This emphasizes the great need for careful attention in the postoperative period to immediate reexpansion of remaining segments and removal of air and fluid from the pleural space.

It might be of interest to interpose a brief comment about other technical matters:

1. In bilateral cases, which side should be treated first? It has been our practice to remove the disease in the side which constitutes the greater burden. If it can be determined by symptoms, x-ray examination or bronchoscopy which side is acting as the principal reservoir for the collection, concentration, and stagnation of secretions, those segments should receive first attention. In a significant number of cases, all symptoms or constitutional effects of the disease disappear or are so minimal that excisional therapy on the side of lesser disease may be indefinitely postponed.

2. It has been our observation that the postoperative management of patients may be complicated after the first single-sided operation. One is confronted with problems of reexpansion on one side while the remaining segments are bathed in secretions from the contralateral septic reservoir through the intercommunicating bronchial system. To solve the problem of the flooding of the remaining healthy segments with material from the contralateral lung in bilateral disease, we have, for some time, toyed with the idea of completing the excision of all abnormal segments, even if bilaterally situated, in one stage.

3. From a group of seventy patients in whom a bilateral operation has been completed, in two instances a one-stage bilateral operation was done. In one other patient (nonbronchiectatic), who suffered from bilateral disease which required excisional therapy, the treatment was completed in one stage.*

(Slide 1.) A 46-year-old woman suffered from bronchiectasis in the inferior subsegment of both anterior segments, the right middle lobe, and the lingula. With the patient in the prone position, the diseased segments on the right side were removed on Jan. 8, 1952. While the wound was being closed, a left thoracotomy was made and the involved segments on that side were removed. The patient's convalescence was prolonged because of the necessity to drain a small localized empyema which developed in an anterior location. Offending organisms were resistant to antimicrobial agents. The hospital stay extended over a period of twenty-six days.

(Slide 2.) A 29-year-old woman had a ten-year history with bronchiectasis in the right middle lobe, and the left basal group, and lingula. On Jan. 26, 1952, the left side was treated first and since every detail of the procedure was expeditiously executed, it was deemed safe and advantageous to resect the middle lobe immediately, and this was done. The convalescence was uneventful, and the patient was discharged on the thirteenth postoperative day.

*Overholt, Richard H., Rheinlander, Harold F., and Bolton, Houck E.: Simultaneous Bilateral Pulmonary Segmental Resection, Bull. New England M. Center **14**: 1, 1952.

(Slide 3.) A 53-year-old man had a survey lesion. An abnormal shadow suggestive of a primary tumor was found in the right upper lobe and a discrete "coin" shadow in the left upper lobe. On Feb. 7, 1952, a right thoracotomy revealed a mass in the contracted posterior segment and, after its removal, a tuberculous lesion was found. While the wound was being closed, a second team opened the left chest where a discrete hamartoma was found which was removed by enucleation. Convalescence was uneventful, and the patient was discharged on the tenth postoperative day.

None of the three patients required tracheal or bronchoscopic aspiration postoperatively. Re-expansion was satisfactory in all cases. None experienced more than the average amount of discomfort. There was no appreciable splinting of the thoracic cage nor was there dyspnea.

THE INDICATIONS FOR AND THE RESULTS OF COMMISSUROTOMY FOR MITRAL STENOSIS

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SELDOM in the history of the medical profession has the introduction of a single surgical procedure so revolutionized the therapeutic demeanor and prognostic thinking toward a common disease entity as has commissurotomy for mitral stenosis. In five short years of pioneer effort, the physical and mental dissolution of the patient suffering from this disastrous and cicatricial end result of rheumatic fever has been revitalized and remodeled to an attitude of hope and assurance. Indeed, the demonstration that the left atrium of the heart can be entered at will to reconstruct surgically the obstructed mitral valve has paved the way for similar approaches to each of the other valves and has led to the establishment of intracardiac surgery as a definitive specialty.

Three years ago, before this association, our initial efforts in the field of valvular surgery were reported.[1] Much skepticism was engendered in the minds of both protagonists and antagonists. The fact that in years gone by a number of attempts to overcome mitral obstruction had terminated fatally in the hands of highly competent surgeons tended to dull and becloud the reception of a possible repeat performance. The demonstration, however, that the release of obstructed valves could be accomplished without the simultaneous production of lethal or even significant valvular insufficiency—originally considered to be the inevitable corollary to such surgery—restored to reality the hopes and dreams of many investigators both living and dead whose original efforts had until recently remained unheralded.

It is the purpose of this article to present the complete data pertaining to our first 256 consecutive commissurotomies for mitral stenosis. Previous communications have reviewed the contributions of the many investigations in this field.[2-9] These reports have also detailed the pathologic and physiologic considerations of this valvular deformity, together with the technique employed for its surgical correction.[10-15] It is, therefore, sufficient to state here that mitral commissurotomy is a procedure in which the individual anatomic leaflets of the stenotic valve are separated. By incising the angles or commissures of the mitral slit its obstructing effect can be overcome and a considerable degree of valve function can be re-established. It is to be

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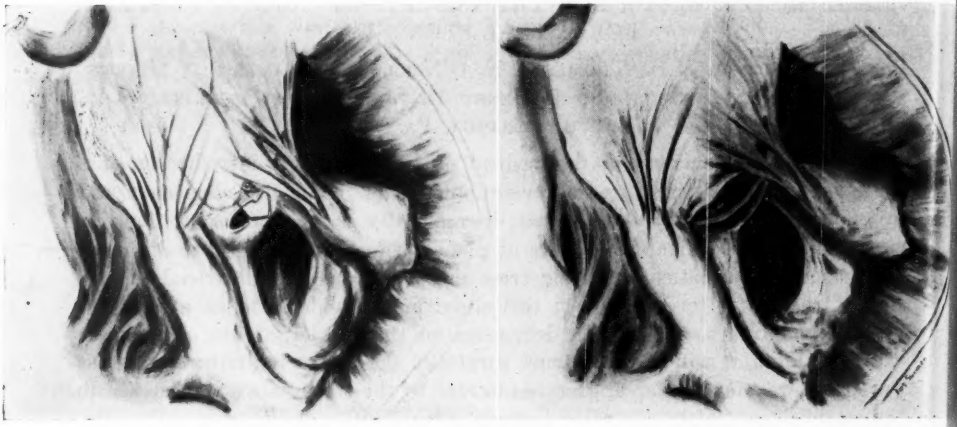
noted that no valve tissue is removed, thus allowing the liberated though thickened and deformed valve leaflets to open during ventricular diastole and approximate during ventricular systole without the production of additional significant regurgitation (Fig. 1).



A. *B.*

Fig. 1.—View of the stenotic mitral valve cone from the left ventricle. *A*, Right index finger and Glover-Avery guillotine knife in position for incision of anterolateral commissure. *B*, Anterolateral commissure completely separated greatly enlarging the orifice with partial restoration of valve function.

ANALYSIS OF CASES

We have performed 256 mitral commissurotomies from Feb. 2, 1949, to April 30, 1952. Fifty-three of these have been performed since Jan. 1, 1952, and except for mortality data are not included in the statistical analysis at this time. Similarly, thirty-nine others were performed for and with other thoracic surgeons on their own services throughout the country by invitation. These will be reported in detail by them at some future time. The operative mortality rate for the entire series was 5.4 per cent, and the over-all mortality, including late deaths, was 7.8 per cent (Fig. 2). These later deaths occurred almost exclusively in stage V cases, over six months after surgery, from a continuation of their irreversible cardiovalvular and pulmonary vascular disease unaffected by surgical intervention.

The body of this report concerns itself with the remaining 164 consecutive cases with a six-month to three and one-half year follow-up period for analysis. A lapse of at least six months is essential before results may be evaluated, for experience has shown that in many instances at least this period of time must pass before the patient can fully appreciate the full subjective benefits of surgery. Actually, a longer period is often required before all objective evidences have become stabilized.

Of these 164 cases, 118 were female patients and 46 were male. The youngest was 4 years of age and the oldest was 59, with an average age of 35 years. Most of these patients were far advanced in the course of their disease and represented the late or terminal stages of mitral stenosis. They have been grouped according to our previously published classification as shown in Table I.

TABLE I. STAGES OF MITRAL STENOSIS

STAGE	NUMBER	DEATHS		MORTALITY %	
		OPERATIVE	LATE	OPERATIVE	LATE
I. Asymptomatic	0	0	0	0	0
II. Statically incapacitating	14	0	0	0	0
III. Progressively incapacitating	105	5	1	4.7	.9
IV. Terminally incapacitating	32	3	0	9.3	0
V. Irreversible	13	2	8	15.3	61.5
Total	164	10	9	6.0	5.4
Over-all Mortality		11.5%			

This classification has been prepared in an effort to combine a functional and therapeutic yardstick for evaluation of the patients for surgery. *Stage I* includes those with the auscultatory findings of mitral stenosis but who as yet have no symptoms. Cases in *stage II* have progressed to the point where symptoms under physical activity have developed, but the patient, living within his

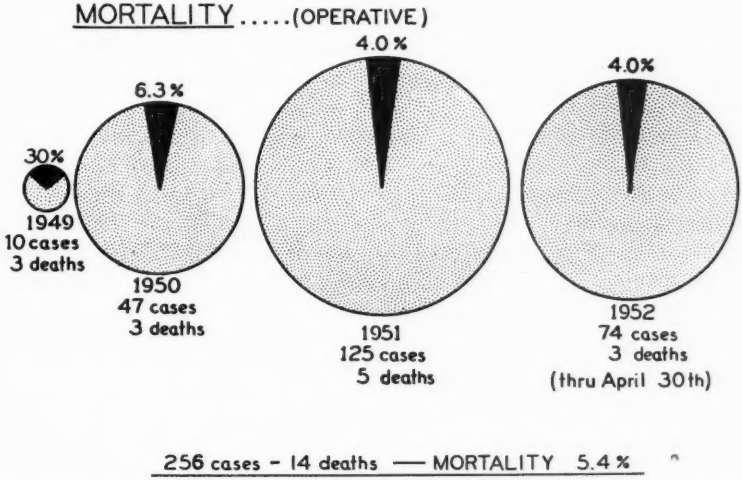


Fig. 2.—Mitral commissurotomy, showing mortality statistics by years from Feb. 2, 1949, through April 30, 1952.

own limitations, remains on an even plateau. *Stage III*, the largest group, and one encompassing many variables, takes in those who, despite the best medical therapy, are losing ground. *Stage IV*, terminally incapacitating, is made up of those patients in whom there is constant evidence of congestive failure even with limited physical activity. Most of these can be rendered relatively free

of their accumulating tissue fluid only by the strictest of medical regimens. A certain small percentage of those in this group will ultimately prove after surgery to have been in *stage V* and to have had irreversible changes. As yet, it has been impossible to separate routinely the patients in these two stages by clinical and physiologic methods; hence, we reserve stage V to classify those whose condition, despite a technically adequate commissurotomy, remains relatively unchanged.

Status Prior to Admission.—Progressive dyspnea and fatigue were present in varying degree in each of the 164 cases (Table II). In addition to this evidence of cardiopulmonary disability, 110 patients had already progressed to the stage of right heart (congestive) failure as evidenced by one or more episodes of hepatomegaly, ascites, and peripheral edema; 59 had had one or more attacks of frank hemoptysis, in several instances to an almost exsanguinating degree. This does not include the many who had expectorated frothy material during bouts of pulmonary edema.

TABLE II. STATUS PRIOR TO ADMISSION

Progressive dyspnea and fatigue	164	100.0%
Congestive heart failure	110	67.1%
Hemoptysis	59	35.9%
Arterial embolism	21	12.8%

In twenty-one patients, cerebral, visceral, or peripheral emboli had occurred on one or more occasions. Of these, fourteen had auricular fibrillation, seven had normal sinus rhythm, and all but six had a history of one or more attacks of congestive failure. It is of considerable interest that only nine of these had gross thrombi in the left auricular appendage at operation. In one additional case the pathologist was able to report the presence of a small thrombus deep in the trabeculae of the excised appendage which was not noted during the commissurotomy. Of greatest significance is the fact that no recurrent embolic episodes have occurred in any of these twenty-one cases (longest three years). It has been previously emphasized that ligation of the base of the left auricular appendage was responsible for this result. However, of equal if not greater importance would seem to be the relief of left atrial stasis accomplished by opening the valve.

TABLE III. ADMISSION FINDINGS

"Pure" mitral stenosis	97	
Mitral stenosis with insufficiency	42	
Mitral stenosis with insufficiency and aortic insufficiency	11	
Mitral stenosis with aortic insufficiency	14	
Left ventricular enlargement (x-ray) (large normal to minimal)	24	
Auricular fibrillation	69	(42%)
Congestive heart failure	43	(26%)
Electrocardiogram: RAD	140	
NAD	23	
LAD	1	

Status Upon Admission.—In the initial phase of this surgical program, every attempt was made to select cases of relatively pure mitral stenosis to insure a proper base line for evaluation of the surgically treated stenotic valve alone (Table III). It has since become apparent that the auscultatory findings of a minimal to moderate systolic mitral and/or aortic systolic or diastolic murmurs in addition to the classical mitral diastolic murmur may be of little consequence provided mitral *stenosis* is the *predominant factor* in the production of the patient's disabling symptoms. Ninety-seven were classified as "pure" mitral stenosis when no regurgitant jet could be felt by the exploring finger at the time of surgery. Prior to this, on purely auscultatory grounds, at least one-third of these had been thought to have associated mitral insufficiency which could not be corroborated at the operating table. It is therefore felt that, contrary to classical teaching, *pure* mitral stenosis can and, not infrequently, does occur without accompanying regurgitation. In forty-two cases, there was an associated minimal (grade 2 systolic murmur) mitral insufficiency, and in seven others there was aortic insufficiency of minimal degree. In fourteen individuals a mitral diastolic rumble and an aortic diastolic murmur were the only murmurs heard. Four others were also complicated by the presence of an aortic systolic murmur of minimal degree.

In the selection of cases for surgery, it is obvious that the careful auscultatory examination by an experienced cardiologist is paramount. The recognition of a typically uncomplicated mitral stenosis by stethoscopic methods presents no problem. The difficulty does not lie with this type of individual but rather with the patient who presents evidence of coexisting mitral stenosis and insufficiency, or multivalvular disease. Here, one must decide whether the mitral stenosis is the predominant lesion. Expert fluoroscopic and radiologic evaluation, with emphasis on individual chamber enlargement, can be of inestimable value in this respect.

One hundred forty of the cases presented a normal left ventricle, and in twenty-four there seemed to be minimal enlargement. In these twenty-four, mitral insufficiency or associated aortic valvular disease was *dynamically* expressing its presence by enlargement of the left ventricle, a finding which we have come to regard as evidence tending to contraindicate surgery. Slight enlargement of the left ventricle, due to mitral insufficiency, is not an absolute contraindication but suggests the possibility of a more equivocal surgical result. If left ventricular enlargement is due to an aortic valvular lesion, commissurotomy might be questioned at this time, although recently two cases of pure mitral and aortic stenosis in which both valves were opened at the same operation have been treated very successfully. The recognition of minor degrees of left ventricular enlargement in the presence of a markedly enlarged right ventricle may well be perplexing. Enlargement of all chambers of the heart is an absolute contraindication.

Auricular fibrillation is not a contraindication to surgery per se unless the ventricular rate is entirely uncontrollable. Sixty-nine patients had auricular fibrillation, and the eventual results have compared quite favorably with those who had sinus rhythm.

Forty-three patients presented themselves in various stages of congestive heart failure, although many others were known to have been in this state prior to admission. In all but thirteen this failure was well controlled before surgery, wherein there existed many years of chronic failure. Except for minimal clinical evidences, these too eventually responded. The period of medical preparation of patients in this status was more prolonged, and in several instances six weeks or more were required. Some of the more dramatic results were achieved in this group after a prolonged period of convalescence.

The importance of hypertension of the pulmonary circulation in the natural history of mitral stenosis has been appreciated for many years. Such classic symptoms as dyspnea, orthopnea, and hemoptysis have been thought to be indications of increased pressure in the pulmonary vascular bed. The roentgenologist and pathologist have supported the clinician's contention of the importance of pulmonary hypertension by describing hypertrophy of the right ventricle, marked enlargement of the left atrium and pulmonary artery, and sclerotic changes in the large and small pulmonary arteries.

In patients with mitral stenosis, right heart catheterizations have recorded high systolic pressures in the right ventricle. Borden and his associates[16] found a rough correlation between the degree of pulmonary artery hypertension and the clinical manifestations. Comparable findings and correlations were observed in forty-five of our first fifty patients in whom catheterization of the right heart was done. The upper limit of the pulmonary artery pressure found in this laboratory in normal subjects is 30/10 mm. Hg. As might be expected, the higher the pressure in the pulmonary artery, the closer the correlation with the clinical and roentgen findings. Usually a systolic pulmonary artery pressure of 50 mm. Hg or greater was associated with a history of prolonged functional disability, episodes of acute pulmonary congestion, hemoptysis, or congestive failure. Such pressures, also, were commonly associated with fluoroscopic and x-ray evidence of marked left atrial and right ventricular enlargement and dilated pulmonary arteries. Electrocardiograms in 140 of this group showed right axis deviation (plus 90 degrees or more), thus reflecting somewhat the degree of pulmonary hypertension.

In a previous communication,[17] the early results of commissurotomy upon the pulmonary arterial pressure have been presented in great detail. In general, it may be stated that the higher the preoperative pressure the greater the postoperative drop, noted particularly in those above 80 mm. Hg systolic in the pulmonary artery. Those ranging between 40 and 80 mm. Hg systolic naturally dropped correspondingly less. The over-all average drop in right heart and pulmonary arterial pressures within one month of surgery was 15 mm. Hg systolic (extremes 0 to 40).

Of much greater interest and significance are the pressure changes which developed and stabilized over many months. One can appreciate the difficulties encountered in obtaining long-range recatheterization data. Although most will and have returned from great distances for postoperative clinical evaluation, few are interested in submitting to a third catheterization (preoperative, early postoperative, and late postoperative). Nevertheless, such

TABLE IV. COMMISSUROTOMY—PRESSURE CHANGES
(mm. Hg—Lilly Manometer)

CASE AND DATE OF OPERATION	PREOPERATIVE		POSTOPERATIVE				MONTHS	RESULTS
			INTERVAL		MOST RECENT			
	RT. VENT.	PUL. ART.	RT. VENT.	PUL. ART.	RT. VENT.	PUL. ART.		
1. J. H. 6/2/50	67/-2 (21)	78/24 (42)	31/4 (13) 1 mo.	30/16 (5)	77/11 (33)	75/29 (44)	5	Unimproved
2. A. N. 10/18/50		(37)		(72)	29/4 (12)	27/14 (18)	9	Excellent
3. G. K. 5/19/50	50/2 (18)	48/24 (32)	31/-2 (9) 1 mo.	36/12 (20)	47/4 (18)	52/23 (33)	10	Improved
4. G. D. 7/6/50	90/0 (30)	84/40 (55)	108/9 (42) 1 mo.	93/48 (63)	50/-2 (19)	50/18 (29)	10	Excellent
5. J. A. 11/8/50	57/-15 (39)	57/24 (35)			45/5 (18)	40/19 (26)	12	Excellent
6. E. M. 1/23/50	55/36 (42)	49/30 (36)	44/0 (15) 1 mo.	45/22 (30)	28/0 (9)	35/10 (18)	13	Improved
7. E. I. 11/9/50	93/6 (35)	105/24 (51)			31/3 (12)	38/13 (21)	14	Excellent
8. M. D. 1/5/51	32/-2 (13)	30/16 (21)			20/8 (12)	20/6 (11)	14	Excellent
9. E. C. 11/3/50	64/-3 (25)	75/27 (43)			30/0 (10)	30/10 (17)	14	Excellent
10. V. S. 6/27/49	141/5 (50)	143/56 (85)	46/-2 (18) 4 mo.	44/13 (23)	33/14 (20)	36/15 (22)	15	Excellent
11. F. L. 7/27/50	68/0 (23)	-	89/8 (35) 7 mo.	89/39 (56)	52/5 (21)	-	16	Excellent
12. J. B. 2/2/49	90/17 (41)	-	45/0 (15) 2 mo.	50/16 (27)	48/3 (18)	-	20	Excellent
13. V. S. 4/11/50	100/9 (39)	130/59 (83)	90/2 (31) 1 mo.	98/37 (57)	26/5 (12)	24/8 (13)	21	Excellent
14. T. S. 11/7/49	43/3 (16)	42/12 (22)			27/5 (12)	-	23	Excellent

data have gradually been obtained in fourteen patients ranging from five to twenty-three months after surgery (Table IV). It is well known that readjustment of the pulmonary vascular circulation, following valvular surgery, may take considerable time, hence the changes noted within the first postoperative month do not necessarily reflect the ultimate pressure or, for that matter, clinical level. Case 11 exemplifies this point. Despite marked improvement in the patient's clinical subjective status at seven months the pressures were significantly higher than those obtained preoperatively. At sixteen months they returned to slightly under the original level representing no essential objective improvement in direct contrast to her very excellent clinical state which permits her to live a normal life, working full time.

Perusal of Table IV will reveal that eleven patients (all but Cases 1, 3, and 11) obtained very satisfactory and significant lowering of the pulmonary and right heart pressures. Surprisingly, in eight of these, the drop was practically to normal levels, representing in several a decrease of 50 to 80 mm. Hg systolic. In each of these eleven the subjective result followed suit so that all are now classified as excellent results by the criteria set forth in the text. One of the remaining three (Case 1) is unimproved clinically after temporary initial relief. Another (Case 3) is improved clinically to a much greater degree than his pressure changes would indicate. The third (Case 11) obtained an excellent result as discussed previously.

The electrocardiogram frequently aids in determining the candidacy of these patients, especially when there is more than one valve defect. Evidence of right ventricular enlargement suggests that the predominant lesion is one of mitral stenosis. It is to be noted that a right axis deviation (plus 90 degrees or more) was present in 140 cases, a normal axis in 23, but more particularly, that in only one instance was there evidence of a left axis shift. In this case, a stage V, mitral insufficiency was found to be equally as prominent as stenosis when the valve was explored. Commissurotomy, although accomplished adequately, had no effect on the course of the irreversible cardiovalvular and pulmonary vascular disease and she continued unimproved until death, ten months later. Finding a left axis shift predicates that other factors are at play and a careful reevaluation must follow.

Operative Findings.—Seldom in the past has there been the opportunity to examine at firsthand the nature of living valvular pathology in situ. It has been a source of wonder to us to find the average stenotic valve in relatively good condition in contrast to the incapacitation of the patient (Table V).

1. The size of the valve orifice, as estimated by the tip of the exploring index finger in 133 of the cases, was approximately 0.5 to 1.0 cm. in diameter or, in more readily appreciated terms, the size of one to two heads of kitchen matches. The cusp margins were thickened and fibrotic with fixation of the periorificial ring. Nevertheless, just enough pliability remained in the valve leaflets so that in only twenty-nine cases could a regurgitant jet of blood be detected with ventricular systole. In the remaining twenty-nine (two were not

TABLE V. OPERATIVE FINDINGS

Size of valve orifice (finger estimation)	
(a) 0.5-1.0 cm. (minimal regurgitant jet in 29)	133
(b) 1.1-1.5 cm. (no jet in 6)	16
(c) 1.6-2.0 cm.	8
(d) 2.5-3.5 cm.	5
	162*
Orifice after commissurotomy	
(a) 4.5 cm.	3
(b) 3.0-4.0 cm.	131
(c) 2.5 cm.	23
(d) 1.5-2.0 cm.	5
	162*
Calcification of valve	75

*Two valves, size not recorded.

recorded) the orifice was somewhat larger. The diameter of sixteen was 1.1 to 1.5 cm., and in six of these no insufficiency could be appreciated. Eight fell into the 1.6 to 2.0 cm. size, three at 2.5 cm., and two were between 3.0 to 3.5 cm. Some of these were obviously poorly selected cases as the result obtained hardly justified surgical intervention.

In contrast to the firm and indurated cusp margins, the valve leaflets, although thickened, were considerably more flexible. Their consistency was usually that of kid glove skin, less frequently like shoe leather. Only twenty had lost all semblance of a functioning valve in that they were completely fixed and rigid. Extreme fibrosis and calcium replacement were responsible for this effect. Thus, as a rule, the "mitral megaphone" could be felt to ride back and forth as a complete cone in response to changes in pressure on its two sides, always maintaining its obstructing effect and unable to open or close at its orifice.

It was possible, despite the pathologic nature of the valve, to accomplish a satisfactory separation of the valve leaflets in 157 cases. Three of these were opened to 4.5 cm. in diameter, 131 between 3.0 and 4.0 cm. (optimum), and 23 others to 2.5 cm. In five the conditions present would permit enlargement to only 1.5 to 2.0 cm. Ideally, the objective of commissurotomy is not only to enlarge the stenotic orifice, but also to restore a considerable measure of valve function. This latter goal could not be attained in approximately twenty-five cases because the valve tissues were replaced by fibrotic scar and calcium to such a degree that all pliability, even after release of the cusps, was lost.

The technique of commissural separation, as used by us, has been repeatedly described in previous writings. Both the right index finger and the specially devised guillotine knife are as a rule introduced simultaneously through an opening near the tip of the purse-stringed left auricular appendage. Whenever possible, after palpation of the entire valve for orientation, one small cut is made across the rind of the anterolateral commissure after which gentle finger pressure is applied. This will usually accomplish dissolution of the commissure to the myocardial wall. At any point where undue

finger pressure is necessary to divide the adherent leaflets, an additional small cut is made facilitating rapid and clean division without the danger of tearing or avulsing either the valve leaflets or their attached chordae tendineae. An anterolateral commissurotomy of this type is, in the majority of instances, sufficient to accomplish maximum results both clinically and physiologically. In a very considerable number of cases one small additional cut was made into the fused and rolled posteromedial commissure just to transect the rigid ring. This was not carried widely into pliable valve tissue, for the danger of producing major regurgitation in this area is great. In the past six months the posteromedial commissure has rarely been opened unless adequate anterolateral separation has been impossible. The over-all result would seem to be better by observing this precaution. When one has become completely familiar with the proper use of the guillotine it will become obvious that its use is essential in perhaps 30 per cent of cases. In an additional 20 to 40 per cent a more complete and thorough commissurotomy can be performed (assuring the patients of a more certain, long-term future) when this technique has been mastered. On the other hand, when thrombotic material is present in the appendage it has been our practice to insert the finger alone, hoping that definitive surgery of an adequate nature can be done by this method in order to obviate the extra manipulation of both knife and finger, lest an embolus from the appendage be produced. Very often, however, the valve has required additional incision and without removing the finger the guillotine is slipped in beside it.

The combination of finger and knife, in the actual division of the commissures, has been used in 114 cases, the knife only in 21, and the finger alone in 28 cases. One was not recorded (Table VI). The anterolateral commissure was divided alone in eighty instances, both the anterolateral and posteromedial in seventy-three, and the posteromedial by itself in four. In six cases only dilatation without separation could be accomplished, in five with the finger and in one with a Kelly hemostat (tiny appendage in a 4-year-old child). One case was not recorded adequately.

2. Calcification was present in some measure in seventy-five of the valves (Table V). The frequent site for such infiltration was on the anterior valve

TABLE VI. TECHNIQUE OF COMMISSUROTOMY—164 CASES

Method		
Finger and knife		114
Knife only		21
Finger only		28
(a) Finger dilatation only	5	
(b) Kelly dilatation only	1	6
		163*
Commisure		
Anterolateral		80
Anterolateral and posteromedial		73
Posteromedial		4
Dilatation		6
		163*

*Technique not recorded, one case.

leaflet over its medial aspect—that portion nearest the outflow tract of the left ventricle. Nineteen of the valves were lightly flecked with tiny calcific beads along the surface of the cusp margins. In forty-four the calcium extended back into the leaflets for varying distances at one or more points of the circumference. Even in these situations considerable mobility could frequently be restored by commissurotomy. In twelve cases calcium was present in larger quantity, practically encircling the mitral orifice and appreciably limiting the reconstructive effect obtainable by incising the commissures. Surprisingly, although little if any valve function could be restored in this group, generous enlargement of the orifice straight through the calcific crust produced an excellent result in four, with considerable improvement in four others. The remaining four were unimproved. Fortunately the anterolateral commissure is less frequently involved by calcium, and separation at this point will improve the patient, as a rule, regardless of the status of the remaining valve cone. It is to be emphasized, therefore, that the presence of calcium per se need not militate against a good functional result. Occasionally, without detectable calcium, the circumferential contraction of the atrioventricular ring, together with extreme thickening of the valve cone and foreshortening of the chordae tendineae, result in such fixation that commissurotomy is to no avail.

3. Gross or microscopic evidence of thrombosis was encountered in the left auricular appendage of thirty-seven patients. Preoperative arterial embolization was associated in only ten of these, and thirty-three were in auricular fibrillation. There was one questionable embolus produced at the time of surgery despite the fact that the finger was introduced through the appendage after proper preparation in each of the thirty-seven cases (see discussion which follows).

Immediate Postoperative Status (Table VII).—

Chest pain: (a) The usual thoracotomy pain, intercostal and chest wall in type, is to be expected and was experienced by all patients.

(b) In addition, pleuropericardial pain may be a striking sequel to this surgery and may be much more severe and persistent than that experienced by the average thoracic patient. It occurred in 38 per cent of cases and may be most prominent during the second postoperative week. Usually it recurs sporadically during the ensuing six months. The pain is typical of that seen in acute pericarditis and similarly is aggravated by swallowing, deep inspira-

TABLE VII. POSTOPERATIVE STATUS—164 CASES

Pleuropericardial pain		63 (38%)
Pericardial effusion		5
Sinus rhythm to auricular fibrillation		23
(a) Transient	8	
(b) Permanent	15	
Auricular fibrillation to sinus rhythm		6
(a) Transient	1	
(b) Permanent	5	
Postoperative hemiplegia		2
Reactivation of rheumatic infection (??)		0

tion, and change of position. The location of the pain may be in the lower substernal area, either localized or radiating to the neck or back. At times a small pleural effusion or early signs of pulmonary congestion may develop.

The importance of this pain lies in its characteristics of persistence, severity, unpredictability, and its partial response to salicylates but not to antibiotics, and ordinary doses of narcotics. In several extreme instances this distress persisted daily for three months, precipitating right heart failure. Such a morbidity is rare and, fortunately, in these instances recovery has been complete and the patients have returned to normal activity. An attempt has been made to anticipate the probable onset of this complication by investigating the patient's rheumatic state prior to surgery. It has been impossible to predict with any certainty such complications, however, either from the history, the physical examination, or laboratory studies.

Pericardial effusion: There were nine instances in which pericardial aspiration seemed desirable. No fluid was obtained in four, less than 100 c.c. in four, and 150 c.c. in one. This type of effusion is not associated with the usual signs and symptoms of cardiac tamponade. The exhibition of undue anxiety associated with a feeling of strangling and oppression should arouse suspicion. The apical rate may be normal or slightly elevated; moderate orthopnea and hyperpnea may develop with profuse perspiration, and as a rule the systolic blood pressure is lowered. The jugular veins infrequently are distended. Even as the fluid is withdrawn the patient may experience subjective relief. Repeated aspiration has not been necessary.

Arrhythmias: Auricular fibrillation developed postoperatively within the first ten days in twenty-three individuals who had had sinus rhythm prior to surgery. Eight were transient and returned to a sinus mechanism with or without quinidine, and fifteen became permanent.

There were six instances in which auricular fibrillation reverted to sinus rhythm after surgery. Only one of these has returned to a state of fibrillation.

Postoperative emboli: Two patients awoke from anesthesia with a hemiplegia. No evidence of gross or microscopic thrombosis was present in the left auricular appendage in either so that the origin of the embolus was unknown, possibly from the valve itself. One-third, in whom the appendage was obliterated by thrombotic material, developed pain in the popliteal area of the left leg shortly after surgery. This patient had had innumerable emboli to the brain, viscera, and extremities in the past, had undergone an attempted embolectomy in the right femoral artery several years before, and had been on Dicumarol for many months prior to admission. Bilateral intermittent claudication had been present for a long time. A therapeutic caudal block was continued for two days without noticeable effect as the left leg became more and more discolored with signs of demarcation below the knee. The right foot was only slightly cyanotic, nonpainful, and maintained unlimited function. Left femoral artery exploration revealed a good but not pulsatile flow of blood and no embolus could be located either above or below from the aortic bifurcation to the pop-

liteal artery. Eventually, a mid-thigh amputation was necessary and the patient made a fine recovery. Extensive search of the vascular tree in the amputated limb failed to discover fresh embolic material, although all vessels were thickened and contracted as in thromboangiitis obliterans.

Reactivation of rheumatic infection: In our considered opinion there has been no instance of an exacerbation of acute rheumatic fever as ordinarily seen in children. This was somewhat unexpected in view of the histologic evidence of the rheumatic state in 49 of the 143 appendages examined microscopically. No doubt a considerably higher incidence of rheumatic stigmas would have been found had the myocardium proper also been examined. This opinion is felt to be true in view of the lack of evidence of carditis exemplified by the development of additional valvular lesions. No change has occurred in the valvular status other than that which was produced traumatically, namely, a mitral systolic blow. In addition, using the major and minor criteria of Jones for the diagnosis of rheumatic fever and its activity, migratory polyarthritis, fever responding to salicylates, subcutaneous nodules, routine increase in the sedimentation rate, and the like have not developed. One outstanding complication is at least under suspicion as being of rheumatic etiology, namely, pleuropericarditis, which has been temporarily present in varying degree during the first six postoperative months in sixty-three patients. Four patients had a continuation of fleeting and migratory bone and muscle aching present for years but this was not made worse or actually affected one way or the other by surgical intervention. The two children in the series did fulfill the generally accepted criteria for rheumatic activity but in all probability were in an active state at the time of surgery. Further observation will be necessary before the role of rheumatic infection as influenced by surgery can be evaluated.

Auscultatory changes: A loud pericardial friction rub was almost invariably heard immediately after surgery, disappearing slowly within ten days.

The mitral diastolic rumble becomes greatly diminished in intensity, and during the first seven to ten days may be inaudible.

In most of the cases a mitral systolic murmur was produced. Its usual intensity may be graded as 1 to 2 and this murmur usually persists. It is felt that as a rule this murmur does not represent a significant degree of insufficiency.

The sharp and accentuated mitral first sound usually remains unchanged in quality, even though the diastolic rumble may disappear. This sound appears to be the least affected by mitral valve surgery. The pulmonic second sound frequently remains accentuated, even though the pressure in the pulmonary artery has been reduced.

Thus, to summarize, the murmurs may change materially but the individual heart sounds may not.

Present Functional Status.—There is no more difficult task in the compilation of clinical data than to reach agreement on suitable and self-explanatory criteria for the evaluation of surgical results. This is especially true when the

pathologic and physiologic pattern of a disease entity, such as mitral stenosis, is so varied. The common ground for comparison would seem to be a functional status of the patient observed at least six months or more after surgery.

Table VIII summarizes the results as obtained in this series. Eighty patients are classified as being excellent in that they have been restored to a normal, productive life, enjoying normal activities without obvious limitation. Limitation of diet and mercurial diuretics are no longer necessary. Fifty-four are still taking digitalis, in most instances, however, because neither physician nor patient has gotten around to discontinuing it. Forty-five are considered to be objectively and subjectively improved, as evidenced by their return to almost normal life and activity within their own particular limitations, determined on a trial and error basis. Most of this group are under medication as a precaution because of the demands of their more greatly enlarged sphere of activity. A few, terminally incapacitated, have now become so amenable to therapy that reasonable activity requires digitalization only. The range of improvement in this group varies widely with the condition of the valve found at surgery and the degree to which valve function could be restored. Their progressive downhill course has been abruptly terminated or reversed, some to regain a high level of efficiency, and others to remain on an improved plateau.

TABLE VIII. PRESENT FUNCTIONAL STATUS*—164 CASES

STATUS	NUMBER	PER CENT	
Excellent	80	48.7	76%
Improved	45	27.4	
Unimproved	20	12.1	
Deaths			
(a) Operative	10	6.0	
(b) Late (8, stage V) (1, stage III)	9	5.4	

Nineteen patients have been essentially unimproved. Seven of these had multivalvular disease with significant mitral insufficiency, the seriousness of which was appreciated only at the time of valve exploration. Psychoneurosis of major degree became more prominent after surgery in four, making cardiac evaluation difficult, although three were considered improved from a circulatory standpoint by their referring physicians. In three there was aneurysmal dilatation of the left atrium, with associated mitral insufficiency. The pathologic nature of three other valves was such that a satisfactory commissurotomy could not be accomplished. The inadvertent production of significant amounts of insufficiency by opening the posteromedial commissure too widely militated against a good result in the final two.

The operative mortality (Table IX) in the 164 cases under complete analysis was 6.0 per cent (ten cases). As might be expected, the deaths all occurred in the more advanced stages of the disease (stages III, IV, and V). In the 105

TABLE IX. CAUSES OF DEATH—164 CASES

OPERATIVE			LATE	
Technical		5	Irreversible cardiovalvular and pulmonary vascular disease with progression into terminal failure. (all in stage V)	8
(a) Split cusps (finger)	2			
(b) Medial commissure				
(1) Cut too far	2			
(2) Unexplained	1			
Patients near extremis		2	Pre-existing insufficiency increased by surgery	1
Cerebral anoxia during surgery		2		
Unsuspected associated aortic stenosis (forward failure)		1		
Totals		10		9

cases in stage III, five deaths occurred (4.7 per cent). For the most part these were due to faulty technique early in this series when experience was minimal. Two were the direct result of the finger pressure method which accounted for the inadvertent splitting of a valve leaflet (one) or partial avulsion of one leaflet from the myocardial wall beyond the commissure (one). In one other there was no anterolateral commissure present and wide incisional separation of the posteromedial commissure produced a lethal insufficiency with the patient dying three weeks after surgery. Hypotension throughout surgery with its attendant cerebral anoxia was responsible for death within two hours of an otherwise satisfactory technical commissurotomy. The fifth death was due to forward failure in a patient who at autopsy proved to have an unsuspected severe aortic stenosis in addition to the mitral stenosis for which she was operated upon.

The cause of three deaths in the stage IV group was not entirely clear. It was thought that one was due again to hypotension and cerebral anoxia as the patient died without reacting in six hours. Post-mortem examination revealed a generalized cerebral edema and nothing else to account for death. The second case was totally unexplainable in a patient who should have done well and in whom autopsy was not permitted. The third was brought about by the surgical production of too much regurgitation from a bilateral commissurotomy in which the posteromedial commissure was opened too widely. It was further complicated by a left hemiplegia.

Both fatalities in the stage V cases were in patients in the extreme terminal state. One was in uncontrolled congestive failure and was done under great pressure. The other required continuous oxygen in the semi-Fowler position, and yet seemed free of clinical decompensation. Examination later uncovered their extensive pulmonary fibrosis and generalized irreversible cardiorenal-hepatic changes.

There were nine additional deaths which occurred from one month to two years after surgery (Table IX). All but one of these were grouped as terminally incapacitated, irretrievable cases by the classification outlined previously. One patient died from multivalvular disease with mitral insufficiency increased by surgery. The other eight, despite an adequately performed valvular operation in keeping with the pathologic conditions encountered, died because irreversible cardiovalvular and pulmonary, hepatic, or renal vascular changes were present, which no mere relief of mechanical obstruction could alter. They rep-

resent failures on the part of an unsuspecting medical profession. At this stage in the development of cardiovalvular surgery they are entirely excusable and may in a sense be regarded as tragic examples with which we all must be re-educated. With a greater understanding of the inherent nature of progressive mitral stenosis and the clear demonstration of a technically successful operation, both as to a low mortality and the satisfactory results obtainable, the selection of patients at an early stage now becomes the paramount duty of every practicing physician and procrastination can no longer be tolerated.

Indications for Commissurotomy.—In the light of the foregoing presentation, the selection of patients for surgical intervention can be simply stated. The ideal candidate is the patient with pure mitral stenosis and beginning symptoms of cardiopulmonary dysfunction such as shortness of breath upon exertion. Fatigue out of all proportion to the patient's physical activity is frequently a prodromal or accompanying finding. At the moment the mere presence of a well-defined mitral diastolic murmur without accompanying symptoms is regarded by many as insufficient reason to suggest surgery. No doubt the day will come in the very near future (if not at present) when such a course will seem unreasonable. No one today awaits the complications and end results of mechanical deformities such as patent ductus arteriosus or coaretation of the aorta before proceeding to their surgical correction. On the contrary, it is the accepted operative practice to proceed shortly after the diagnosis has been made clear and long before symptoms and signs of irreversible damage have supervened. Surgery for mitral stenosis at such an early stage would carry an infinitely lower mortality than it does today and much cardiac disability might be obviated.[18, 19]

All other indications are merely compromises from the ideal, but for years to come an understanding of the more advanced but altogether salvable states is essential. An outline under seven major categories (and contraindications) will most succinctly serve this purpose:

1. History
   (a) Early cardiopulmonary dysfunction—ideal
   (b) Marked dyspnea, hemoptysis, reversible failure—acceptable
2. Age—elastic range, physiological rather than chronological
3. Valvular defect
   (a) Pure mitral stenosis—ideal
   (b) Associated mitral insufficiency and/or aortic valve lesion in presence of normal left ventricle—acceptable
4. Roentgenogram
   (a) Left atrium and right ventricle minimal enlargement—ideal
   (b) Minimal left ventricle enlargement—questionable but acceptable
5. Electrocardiogram
   (a) Normal electrical axis or right ventricular strain
   (b) *Never* left axis shift
   (c) Auricular fibrillation with controllable ventricular response—acceptable
6. Functional capacity
   (a) Stages I and II, statically incapacitating at the most—ideal
   (b) Stage III, progressively incapacitating—acceptable
   (c) Stages IV and V, debatable—occasional good result

7. Complicating factors
   - (a) Arterial embolic episodes
   - (b) Recurrent hemoptysis
   - (c) Hypertension (?)

   } Acceptable, and may be urgent
8. Contraindications
   - (a) Acute rheumatic fever
   - (b) Subacute bacterial endocarditis until controlled
   - (c) Associated marked mitral insufficiency or aortic lesions with all cardiac chambers enlarged

In conclusion, surgical reconstruction of the stenotic mitral valve is a major adjunct and an absolute essential in the continuing care of these rheumatic victims. Although such patients can and have been restored to a high level of efficiency and, as such, enjoy a more normal life, nevertheless, if referred for surgery late in the course of their disease, they must always be considered as cardiac patients under the watchful care of their attending physicians just as the surgically treated tuberculous patient will require frequent observation, advice, and management. The burden of selecting patients at an early stage rests squarely on the shoulders of both physician and surgeon and this responsibility can no longer be avoided or postponed.

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DISCUSSION

DR. CONRAD LAM, Detroit.—I wish to congratulate Dr. Glover and his group on continuing the work begun with Dr. Charles Bailey. I should like to discuss his paper by presenting a very small contribution to technique which we have found helpful and which will, I believe, affect the results.

(Slide.) This is what we call a six-fingered commissurotomy glove. Those who have done commissurotomies by the technique of the authors of the preceding paper realize that there is sometimes considerable hemorrhage. Blood flows between the two gloves along the knife and escapes through the slit in the outer glove. At the suggestion of Dr. Edward Munnell, we have added to the ordinary glove a sixth finger on the palmar surface through which the knife can be passed and around which a tight ligature can be placed. Then the surgeon can take his time, deciding whether or not to make a cut here or there. Actually, the knife can be rotated until its flat surface is perpendicular to the finger and then the cutting edge can be palpated. This is particularly helpful in incising the medial commissure, when orientation is especially important.

(Slide.) This photograph taken during a commissurotomy shows the finger in the opening in the auricle, hemostasis being maintained by the snare. This is the palm of the glove which is filled with blood, and not a drop escapes.

(Slide.) The construction of the glove is simple. Details will be reported by Dr. Munnell in an early issue of the JOURNAL OF THORACIC SURGERY.*

I should like to mention one embolic episode in an otherwise uneventful commissurotomy. When the patient was being transferred to the stretcher, it was noted that both legs were cadaveric in appearance. The patient was lifted back onto the table, the instruments were quickly sterilized and, through a retroperitoneal approach, a saddle embolus the size of a small egg was removed. The patient now has normal circulation in the legs.

DR. CHARLES P. BAILEY, Philadelphia.—I should like to present a common complication in surgery for mitral stenosis.

(Slide.) This is a picture of the brain destruction following arterial embolization. This nearly always involves the middle cerebral artery when it occurs during commissurotomy, or I guess at any other time. You can see why these patients have residual paralysis of one side if they survive—why they have a degree of aphasia if this damage affects the left side of the brain.

(Slide.) This is a diagram representing the insertion of a finger through a clotted appendage, dislodging particles which can cause peripheral arterial embolization. Approximately 30 per cent of the patients who come to commissurotomy have some degree of thrombosis in the appendage, so that this accident is not completely preventable in all cases.

(Slide.) This is the other major source of arterial embolization—calcium deposits on the edge of the valve which may be inadvertantly dislodged at the time of cutting, or of breaking the commissures with the finger. Often this calcium is like loosely attached sand; it rolls under your finger and you know you have released small particles into the circulation.

Entrance into the heart by way of the left superior pulmonary vein can be made. I have made them in about twenty-three instances. However, two patients died as a result of that technique and so it seems to be more dangerous than embolization to the brain itself. In a personal series which I worked out last November there were twelve cases of cerebral

*Munnell, Edward R.: Six Fingered Glove for Mitral Commissurotomy, J. THORACIC SURG. **23**: 628, 1952.

embolization in 235 operations, an incidence of about 5 per cent. Eight of those patients died, mainly as a result of that embolization. Since November, 1951, a series of 157 patients have been operated upon by a technique with modifications as shown.

(Slide.) This represents the circulation to the brain and you can see that the middle cerebral arteries, which are the site of most of the emboli, are direct continuations of the internal carotid arteries. Obviously, if you could obstruct them during the course of the intracardiac surgery you would not have any trouble with cerebral embolization. In my entire experience with approximately 500 cases I have had only one case of embolization to the lower extremities during commissurotomy, so it is obvious that the little emboli that undoubtedly get to the vessels of the lower extremities do not necessarily cause any trouble. The main problem is to protect the brain.

(Slide.) This is what we have worked out. It may not be the best way, but it is the way we did it. We mobilized the left common carotid artery in the mediastinum above the aortic arch, and placed a piece of umbilical tape around it. Then at any time in the surgery the umbilical tape can be pulled up, kinking the vessel so that no blood flow occurs. We think that this can be done for at least three minutes, while the right innominate artery is completely blocked, without causing any cerebral damage. Since the mediastinal portion of the innominate artery does not lend itself well to kinking, we had to adopt a more definitive technique of management. A bulldog clamp is placed across the origin of the innominate artery at the beginning of intracardiac surgery and is left on throughout that portion of the surgery, in one case, 34 minutes. I should like to report that we have had no complications while using this technique, but that is not quite true. In spite of using this technique in 157 consecutive patients, we did have one case of left-sided cerebral embolization. I suppose a clot may have lodged there either between our periods of traction upon the umbilical tape, or perhaps after we were through. We thought we were adequately protecting the brain by traction upon the umbilical tape whenever we were doing anything inside the heart and for fifteen heartbeats thereafter, but this patient still developed a left-sided embolus.

We also had one other case with a cerebral complication, although I do not believe it was due to an embolus. This man previously had had a right-sided cerebral embolus and he had a left auricle almost completely full of clot. We were so afraid of a repetition of the embolus that we made longer traction on the left common carotid perhaps than we should have, about five minutes. With the previously diminished right cerebral circulation, and the innominate being clamped off, he has developed an exaggeration of the previous paralysis on the left side. Therefore I think we may have to limit our time of carotid obstruction more strictly.

DR. HENRY SWAN, Denver.—I cannot resist paying tribute to the wonderful work which Dr. Glover and Dr. Bailey have done in pioneering this operation for mitral stenosis. I should like to make one brief comment concerning the selection of patients. Dr. Glover has ably demonstrated that operation can be done at low risk and certainly should be done early in the course of this disease. It is generally agreed, I believe, among pathologists, that in the presence of long-standing venous and associated arterial pulmonary obstruction, changes occur in the pulmonary vascular bed which eventually become irreversible and which maintain, of themselves, the pulmonary hypertension. In the course of mitral stenosis, therefore, there comes a time when operation can be done only at a much increased risk and without promise of much benefit to the patient. My colleague, Dr. Blount, and I have been interested in studying this phase of the problem, and have been doing so by attempting to evaluate the response of the pulmonary circulation to exercise. In the normal individual who exercises, of course, the pulmonary artery pressure does not rise materially and the cardiac index goes up sharply. In the patient with mitral stenosis the pulmonary artery pressure, which may be slightly or moderately elevated at rest, on exercise shows a very pronounced rise and the cardiac output does not show a very great increment. This rise in pulmonary arterial pressure would appear to be the result of two components. The first is the obstruction at the venous end, that is to say, the obstruction of the valve itself; this can be measured by the use of the so-called "pulmonary capillary pressure." The other component

is the resistance of the pulmonary vascular bed itself; this can be measured by subtracting the pulmonary capillary pressure from the pulmonary artery pressure. It appears likely that in most patients who have a pulmonary artery pressure rise due primarily to and associated with a marked rise in pulmonary capillary pressure, the valve itself is causing the pulmonary artery rise and operation would be of benefit. On the other hand, if a marked pulmonary artery pressure occurs, a rise in pressure which is not associated with a pulmonary capillary pressure rise, then it seems reasonable to assume that the peripheral resistance in the vascular bed of the lung itself is causing this rise, and that operation on the valve would would not be of great benefit to this patient.

We have doing lung biopsies at the time of operation in the attempt to correlate these physiologic observations with the observed changes in the pulmonary arterial tree. It is still too early for us to be able to say whether this concept has validity, but I believe that if so, it may be of some help in the selection of patients and in the prognosis of what benefit may be derived from the operation.

DR. HARRISON BLACK, Boston.—I should like to congratulate Dr. Glover on his excellent series and reported gross mortality of 7 per cent. However, we on Dr. Dwight E. Harken's services at the Peter Bent Brigham, Boston City, and Mt. Auburn Hospitals are impressed with the advisability of a breakdown of mortality statistics. Dr. Glover's results are certainly commendable in the aggregate. However, to tell certain patients that they have seven chances in 100 of dying as a result of the operation would be excessively pessimistic. On the other hand, some patients have nowhere near 93 chances in 100 of surviving. Mortality statistics should serve to tell us how well or how badly we are doing in any particular group of patients so that we may make intelligent decisions in the choice of patients for operation. Patients who have mitral stenosis range the spectrum of illness from no disability in athletes to those who succumb to their disease. The results we get in survival and rehabilitation depend on the part of the spectrum in which we are working, what we do in this area, and how we take care of the patients before and after operation.

Obviously, the starting point for such an analysis is a classification. (Slide.) All patients with mitral stenosis that we see are placed in one of four clinical groups. This is done preoperatively without regard to factors that might increase the operative risk, such as calcified auricular thrombus, previous history of peripheral or pulmonary emboli, calcification of the mitral valve, or the existence of other valvular lesions.

Group I is the one we consider *benign*. In it are placed all patients who have the murmur of mitral stenosis but who are not disabled by it. Because many of these people have a good prognosis and may lead an active and useful life we do not advise them to have an operation. They must, however, be followed carefully lest they slip into a more advanced group.

Group II consists of those who have various degrees of incapacity, predominantly due to dyspnea, but who are getting no worse. In this *disabled* group we have only recently accepted patients for operation, for we previously felt that they were functioning adequately enough to wait until our technique had become stable. It has now done so. To some of the patients in this group whose incapacity interferes with their ability to earn a living or to live a life acceptable to them, we are now offering mitral valvuloplasty.

It is in Group III, the one we call *hazardous* because of the poor prognosis on medical management, that we feel the operation has the most to offer in terms of rehabilitation at an operative risk that now compares favorably with that of any major abdominal procedure. This is the group whose dyspnea and limitation are progressing. Many of them have had repeated episodes of pulmonary edema brought on by the slightest exertion or excitement; many have had hemoptyses or emboli; a large percentage of them have fibrillation; and they are generally on digitalis, limited salt intake, and mercurial diuretics. In spite of the best of medical management they are becoming more disabled. It is important to have this clinical picture in mind when we consider the mortality figures, for these patients are well down on the scale of severity of disease.

It happens that we have operated on a large number of patients in Group IV. This is the group of *terminal* patients who are rapidly failing, frequently are in irreversible failure, and have considerable damage to myocardium, lungs, and liver. Many of them are able to exist only at a bed-and-chair level of activity.

(Slide.) In this slide is illustrated the breakdown of mortality according to the groups as outlined. As you can see, there have been 257 total operations for mitral stenosis in this clinic. Many of the earlier procedures have now been abandoned. Eliminating these there have been 242 finger fracture valvuloplasties. In Group I there have been no operations. In Group II, the handicapped but not progressive cases, there have been 14 operations with no deaths. In Group III, the hazardous group, we have done 130 valvuloplasties up to the present time with six operative deaths and no late deaths, giving a mortality, operative and late, of 4.6 per cent. In the last 100 patients in this group there have been only two deaths, one in a patient with a solidly calcified auricular clot and another apparently from adrenal exhaustion.

In considering the terminal Group IV patients one can appreciate how desperately ill they are from the medical mortality. Early in our experience we had the opportunity to observe 19 Group IV patients who were classified by exactly the same criteria as the others but who refused operation. Seventeen of these died within a year, 15 within six months, giving a medical mortality of 89 per cent. Another fact worth mentioning is that in a few instances we have felt that a patient was too ill to accept for operation. None of these has survived for more than a week after this decision. In Group IV patients we have done 98 valvuloplasties with an over-all mortality, operative and late, of 31 per cent. Expressed another way, we have been able to salvage 69 per cent. In the last 45 procedures in this group the mortality has been reduced to 20 per cent.

The point of all this is to demonstrate the marked contrast in risk and salvageability in the various stages of the disease, mitral stenosis. Obviously it is not best to operate in the terminal group; we do not like to do so. We are, however, forced to it quite often because some internists wait until the patient gets into this desperate condition. It is imperative that we educate our medical colleagues to refer these patients for operation at a time when there is a reasonable chance to do something for them. It can no longer be said that the operative risk justifies a policy of watch and wait when the patient is obviously getting progressively worse.

Just a word about the technical problems of management that face us at present. We can agree with Dr. Bailey that the *bête noire* of mitral stenosis surgery now is the production of emboli either of blood clot or calcium particles from the valve at the time of the intracardiac maneuver. We have not obstructed the carotid arteries in the chest but have had the anesthetist obstruct them in the neck. I do not have the figures, but believe that the embolization problem has been somewhat improved by that method. We have also allowed a brisk flushing of blood out of the auricle before insterting the finger as suggested to us by Dr. John Jones, and in several instances have washed out large clots in this way.

In the Group IV patients the problems are no longer associated with the operative technique. We have reached the era, as I believe most everybody has, in which we can operate with safety and few technical errors. The problems are those of management of the pre- and postoperative fluid and electrolyte balance, management of arrhythmias, and the conduct of the anesthesia. Many of these patients have severe sodium depletion because of long periods of restricted sodium intake and mercurial diuresis on frequent occasions, and some of the Group IV patients have actually died of intractable electrolyte imbalance while attempts were being made to prepare them for operation.

In the field of anesthesia we feel that the efforts of Dr. William Derrick in keeping the patients adequately ventilated with his respiratory assister, and also in using a minimal amount of anesthetic agent, have unquestionably allowed us to operate successfully on patients who would have died with any other anesthetic techniques.

In the postoperative period the Group IV patients often present a severe form of electrolyte imbalance which is a marked exaggeration of the normal response to operative trauma. We have frequently found that the serum sodium level falls to 120 to 125 milliequivalents per liter, and on some occasions we have found values as low as 108. Not all these patients show clinical evidence of the low sodium syndrome, such as persistent hypotension, weakness, and apathy, and complete balance studies in one patient have shown that these low serum figures are not necessarily a true reflection of the total body sodium. However, when the clinical syndrome is associated with a low serum sodium value, we have restored these patients to compensation by the administration of hypertonic saline. We have been immeasurably aided in our management of this problem by Dr. Francis D. Moore and his laboratory associates. On other occasions evidence of adrenal exhaustion has been present and corrected with ACTH or cortisone.

One further point requires comment. Dr. Glover has said that 75 per cent of the patients in his series are improved postoperatively. Our experience has been somewhat different, for in our series no surviving patient has failed to be improved, some of them to a miraculous degree. We feel that when mitral stenosis is severe and is adequately corrected the patient will manifest at least some improvement despite damage to liver, lungs, and myocardium if he survives. Admittedly this improvement may be less than if he were operated upon before "the horse is stolen." We take no personal credit for this; it is probably a matter of diagnostic precision in the selection of patients. For this excellent cardiac collaboration we express our appreciation to Drs. Laurence B. Ellis, Lewis Dexter, and Samuel A. Levine.

DR. DENTON A. COOLEY, Houston.—In our experience at Baylor University with thirty commissurotomies we have had one death. I would like to cite one case which illustrates the application of commissurotomy during pregnancy. This patient was 28 years old and in the fourth month of her first pregnancy. During the past four years she had developed progressive signs of incapacity and had several bouts of pulmonary congestion consequent to mitral stenosis. Commissurotomy was performed on Nov. 2, 1951, and she recovered uneventfully. She is expected to deliver in the next few days, and I understand she has remained active throughout pregnancy. (On May 18, 1952, after a six-hour labor she delivered a 7 pound 3 ounce female infant, and the postpartum course was uneventful.)

At the time of operation I knew of no other such experience, but Mr. Laurence Whytehead reports to me that Mr. Brock of London has treated one case similarly, and I understand Dr. Rumel of Salt Lake City has another. If the combination of pregnancy and mitral stenosis becomes an indication for commissurotomy, I suggest that it be done before the fifth or sixth month, after which the demand upon the left ventricle increases sharply with increase of the placental circulation.

DR. F. E. TRICERRI, Buenos Aires, Argentina.—I should like to report my experience in Argentina in twenty cases of mitral stenosis. Of those, fifteen belonged to Group III, of Harken classification, and there were no deaths in those. In five cases that belonged to Group IV, there were three deaths, due to embolism in the cerebrum. I would like to ask Dr. Glover how to avoid cerebral emoblism. Of those three patients one had five previous embolisms, one had two, and the third had one. In all three cases the appendage was full of blood clots and to wash it out I let blood through the appendage before introducing my finger; I was unsuccessful in coping with the emboli which finally caused death, one in seventy-two hours, one forty-eight hours, and the other within ten days.

DR. GLOVER, (Closing).—I am very grateful to the discussers for their comments. I should like to refer to each in turn if time permitted, but I have been asked to show a strip of film depicting the intracardiac portion of a mitral commissurotomy which would perhaps be of more interest. In passing, let me take exception to the use of the word "benign" in Dr. Black's classification. The word is misleading for in most instances the condition will not remain benign for long and therefore tends to convey a wrong impression.

(Movie.) Many people have asked, "What goes on inside the heart?" They all know how to get to the appendage, so we have prepared this heart specimen of a 24-year-old girl

who came into the hospital during her first attack of pulmonary edema and died within twenty-four hours before surgery could be undertaken. This is the size of the stenotic valve orifice most frequently seen. It is tiny, as you see, and will just admit the head of a paper match. The patient was in extremis, of course, when first seen, and we show this primarily to illustrate that even in a terminal state the valve tissues are often quite pliable. This is the anterior or aortic cusp and here you see the posterior leaflet. They are not the thin cellophane-like normal structures but thickened like the skin of a kid glove. There is plenty of pliability provided an adequate commissurotomy can be accomplished.

The finger is inserted through an opening in the tip of the appendage and the valve and its orifice are explored. In a few seconds the pliability of the valve can be ascertained, where the calcium, if any, lies, and where the commissures are. The anterolateral commissure lies at about 11:00 o'clock, the posteromedial at about 5:00 o'clock. The George P. Pilling Son Company and, more recently, Bruno Richter, have been most helpful in developing these guillotine knives. This demonstrates the method of cutting or starting the cut in the anterolateral commissure, and this particular knife can be turned over and used for the posteromedial commissure as well. Usually the knife will be used with the curve the other way and the anterolateral commissure just opened. One little nick is taken in the commissural angle. Here you see what tremendous improvement just one small cut will make. Frequently, if finger pressure alone is relied upon, all that part of the valve lying parallel to the wall of the ventricle will be avoided or missed, so that we prefer to have a hooked blade available at all times. You see now what a marked opening can be produced by just the anterolateral commissurotomy. One small cut may be made on the posteromedial commissure in this fashion and a little additional opening obtained, but we do not carry the cut on this side very far, for otherwise one might incise into the outflow tract of the left ventricle (the intracardiac portion of the aorta) and thereby produce excessive mitral regurgitation.

NEW INSTRUMENT FOR CARDIAC VALVULAR COMMISSUROTOMY

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THE surgical attack on aortic stenosis has long frustrated the cardiovascular surgeon. Transaortic, including transcarotid approaches, have met with significant failure. Earlier attempts via the left ventricle have resulted in some catastrophes due to unguided and blind techniques.

A successful new instrument consisting of three main features is presented.† It contains, first, a beaded guidewire; second, an expanding or dilating triradiate mechanism for aortic commissurotomy; third, an operating control mechanism.

First, an estimate of the external diameter of the aortic valve ring is made by manual palpation. The instrument's control mechanism is then accurately set, so that the open diameter of the dilator will be slightly less than that of the aortic ring. This prohibits overstretching, tearing, or cracking of the aorta at the time the aortic valve commissures are opened.

By passing the beaded wire into the left ventricle, through a small incision under purse-string control, and up into the stenosed aortic valve orifice, a positive guide is then in place as a path for the dilator to follow. This eliminates time-consuming and injurious probing. The triradiate arms of the dilator are wedge-shaped, as seen on cross section, to allow a leading fin on each arm to initiate the parting of the commissures along their fused planes. The dilating head of the instrument is set on a slight swivel, so that it becomes self-aligned to meet the commissures accurately, as the head is passed into the valve orifice. The dilating action is one of commissure parting, not tearing, or cutting. The stretching action at the mid-portion of the leaflet, which can result in fatal tear, is minimized because of the parting action at the commissures.

The anatomic position of the anterior commissure of the aortic valve is nearly always constant, and an external position overlying this point is readily identified in relation to the take-off of the coronary arteries. Thus, in the original passage of the instrument through the ventricle, the anterior dilating arm of the dilator is positioned roughly, but accurately, by merely turning the handle and shaft a few degrees or, as we would say, "lining it up" with the point in question. The swivel feature of the dilating mechanism then accomplishes accurate alignment of the dilator as it fits into the orifice.

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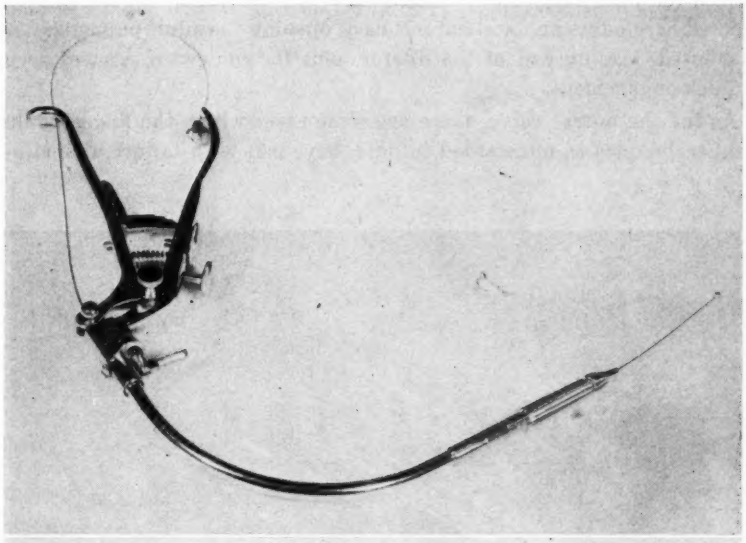
†We are greatly indebted to Major John Shearman Donaldson for the mechanical design and production of this instrument. It may be purchased through George P. Pilling Co., Philadelphia, Pa.

A safety device prohibits opening of the dilator upon entering and withdrawing of the instrument through the ventricular wall.

The position of the acting head of the mechanism is assured by external palpation at the root of the aorta. To date, twenty-three patients have been operated upon, thirteen of the operations being combined with mitral com-

Fig. 1.



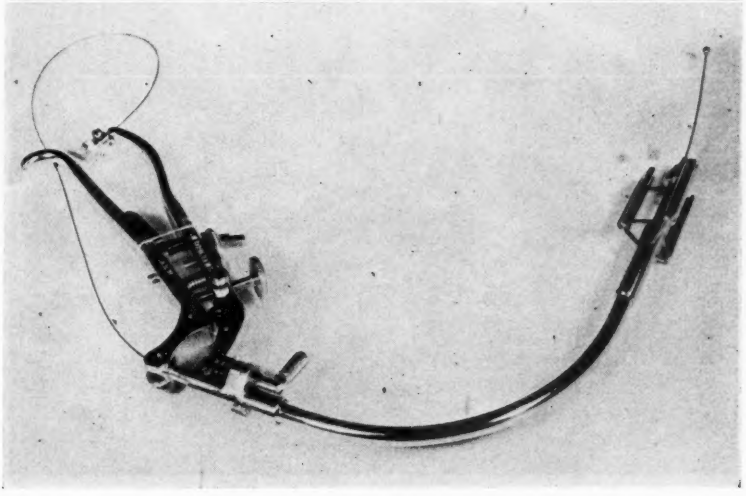


Fig. 2.

Fig. 1.—Aortic dilator (closed.)
Fig. 2.—Aortic dilator (open).

missurotomy, done at the same operation. There has been one operative mortality.

The simplicity of action and the results of this instrument have given us great hope for a more complete attack on pulmonary and mitral valve stenoses. A biradiate dilator attachment has been developed for these two valves, being applicable chiefly in adult cases. The Potts dilator and the Brock dilator are good for the pulmonary stenosis of infants and children, and the instrument presented here offers an excellent means of opening the adult pulmonary valve. The pointed, leading end of the dilator, plus its guidewire, assures accurate and quick engagement.

As for the mitral valve, there are some cases where the finger or instrumental techniques as understood to date, have met with failure, due either to

Fig. 3.

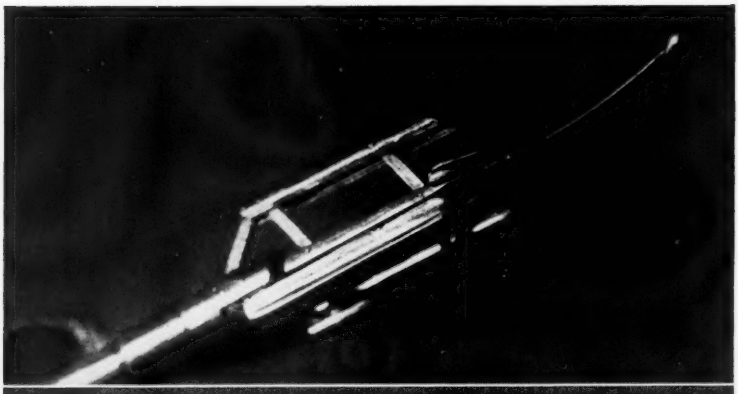


Fig. 4.

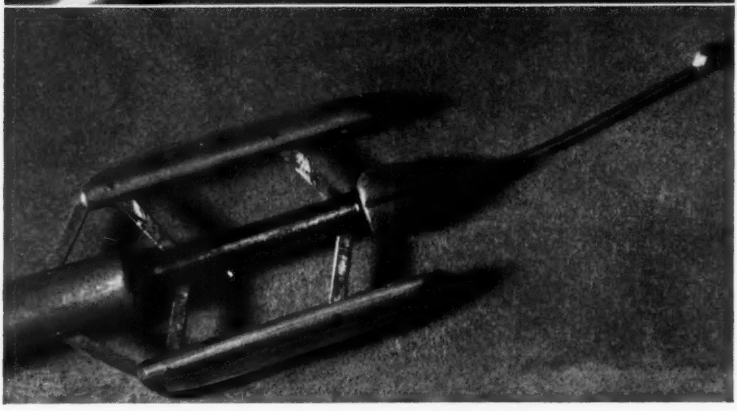


Fig. 3.—Triradiate dilator head with guidewire.
Fig. 4.—Biradiate dilator head with guidewire for pulmonary and mitral stenoses.

the thickness of the valve, presence of a tremendous interauricular thrombosis, or a contracted, tiny, inaccessible auricular appendage. In these, biradiate dilatation is offered as a real improvement in mode of attack. Here again, this improvement is noted because the dilating bar is designed to part the commissure, rather than cut or tear it. Also, there is better control in the actual degree or length of opening which can be accomplished.

At present, the instrument is not being used on thin valvular stenoses, as there is a possibility of false splitting of the septal leaflet in mitral stenosis of this type.

PULMONARY RESECTION IN THE TREATMENT OF LUNG TUBERCULOSIS

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THE principle of resection of tuberculous tissue has recently been accepted in the treatment of pulmonary tuberculosis. With the pulmonary resection, only the active focus of tuberculosis is removed, but it is evident that there are quiescent foci spread all over the lung. Following the operation, there can be reactivation of the disease in the remainder of the lung, or dissemination of tuberculosis in new foci.

After the publications of Churchill and Klopstock,[1] Overholt,[3] and others, there was great enthusiasm for this proceduce. However it was pointed out that the results of thoracoplasty were far superior to those of resection; pulmonary resections were followed by more complications, although they were effective in many cases which could not be satisfactorily treated by any other procedure. Naturally, the complications are directly related to the type of lesions for which the operation is indicated.

Sweet,[5] reviewing 63 cases followed over a period of two years, verified that 28 patients (44.4 per cent) had died, 9 showed active lesions (14.3 per cent), and only 26 (41.3 per cent) were clinically well and had negative tests. These patients, who were submitted to 27 lobectomies and 36 pneumonectomies, were operated on before the systematic use of streptomycin.

Overholt[4] demonstrated the influence of anesthesia, of decubitus during the operation, and of corrective thoracoplasty on the results of lung resection in tuberculosis. Jones and Robinson[2] assembled statistics covering operations performed before 1947 from the records of three large surgical clinics. From a total of 343 cases, the morbidity rate was 51 per cent and the mortality rate 28 per cent. These authors, in a series of 123 pulmonary resections, reported complications in 22 per cent of the cases and a mortality of 12.2 per cent. Among 80 patients receiving streptomycin, the morbidity was 12.5 per cent and the mortality 5.7 per cent.

CLINICAL MATERIAL

This report deals with the results of resection for pulmonary tuberculosis observed in a series of 103 patients, performed from February, 1946, through January, 1951. The general statistical data on these patients are shown in Table I.

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TABLE I. GENERAL STATISTICS (PERIOD BETWEEN FEB., 1946, AND JAN., 1951) ON PULMONARY RESECTION IN TUBERCULOSIS

1. Number of patients			103
2. Number of resections			103
Pneumonectomies		53	
Right	25		
Left	28		
Lobectomies		49	
Right upper	24		
Right upper and middle	1		
Right upper and superior Division of right lower	3		
Right lower	6		
Right lower and middle	2		
Left upper	11		
Left lower	2		
Segmental resection		1	
			103
3. Age of patients (15 to 50 yr.)			
15 to 20 yr.	15		
21 to 30 yr.	53		
31 to 40 yr.	24		
41 to 50 yr.	11		
			103
4. Sex			
Males	42		
Females	61		
			103
5. Duration of disease			
Under 1 yr.	32		
1 to 2 yr.	40		
2 to 3 yr.	14		
3 to 4 yr.	4		
4 to 5 yr.	3		
Over 5 yr.	10		
			103
6. Race			
White	89		
Black	9		
Yellow	5		
			103
7. Incidence of positive sputum	96 (93.2%)		

The cases were thus distributed: 53 pneumonectomies, 49 lobectomies, and 1 segmental resection. In 3 cases, we performed at the same time a lobectomy and a segmental resection in another lobe. The pneumonectomies were done on the right side in 25 instances and on the left side in 28 instances.

The lobectomies were as follows:

24 excisions of the right upper lobe
1 excision of both right upper lobe and middle lobe
3 of the right upper lobe and apical segment of the right lower lobe
6 of the right lower lobe
1 of both the right lower lobe and the middle lobe
11 of the left upper lobe
2 of the left lower lobe

In one case, the apicoposterior segment of the left upper lobe was excised.

The youngest patient of this group was 15 years old, and the oldest, 50 years old. The majority of the patients were in the 21-to-30 group; 61 were women and 42 men. Thirty-two per cent of the patients were sick for less than one year. Of the patients, 96 (93.2 per cent) had positive sputum and 7 (6.8 per cent) were negative before the operation. These patients had bronchial stenosis, bronchiectasis, and "filled" tuberculous cavities.

INDICATIONS

In the beginning of this series, our indications were more conservative, choosing the cases in which the operation could offer a lower risk. However, we soon realized that a great number of patients with severe pulmonary lesions could be cured only through pulmonary resection; these patients presented more complications and fewer cures.

The indications under which our patients were operated on are summarized in Table II.

TABLE II. INDICATIONS IN ONE HUNDRED THREE PULMONARY RESECTIONS IN TUBERCULOSIS

1. Persistent lesions under collapse therapy		41
A. Cavity under pneumothorax	14	
B. Exudative lesions under pneumothorax	11	
C. Cavity under extrapleural pneumothorax	4	
D. Postthoracoplasty uncontrolled disease	12	
2. Large cavities of the upper lobe, with evidence of bronchial disease		16
3. Lower lobe cavities (1 case associated with suppurative disease)		10
4. Extensive unilateral tuberculosis*		24
5. Fibrosis, bronchiectasis, and tuberculous tracheobronchitis†		4
6. Upper lobe disease (suitable for thoracoplasty)		3
7. Tuberculoma or "filled" cavities		4
8. Tuberculosis of a single pulmonary segment		1
Total		103

*One patient with bronchialstenosis and negative sputum.
†Two patients with negative sputum.

In the first group are 41 persistent lesions under collapsotherapy. Among them, there are 14 cavities under total pneumothorax and phrenicotripsia; 11 exudative lesions (caseous pneumonia) under pneumothorax and phrenicotripsia; 4 cavities under extrapleural pneumothorax, and 12 residual cavities after pneumothorax.

In the second group are 16 cases of large cavities of the upper lobe, with evident signs of bronchial tuberculosis; many of these cases represented an actual disappearance of the upper lobe.

In a third group are 10 cases of cavities of the lower lobe, all of them persistent after phrenicotripsia.

The fourth group concerns 24 cases of extensive lesions, multilocular, localized in a single lung.

In the fifth group are 4 cases of specific bronchiectasis, bronchial tuberculosis, and more or less pronounced fibrosis of the lung.

In the sixth group we included three cases of excavated upper lobitis; in these cases we had to determine whether the indications were for lobectomy or thoracoplasty.

In the seventh group are 4 cases of cavities with a closed bronchus, "filled" with organizing caseous material. These cases are similar to the cases described as tuberculomas by the American authors.[2]

The eighth group considers a patient in whom the lesions were restricted to a segment of the lung. In this case a segmental resection was performed.

Some of these indications, such as lesions under thoracoplasty, insufflated lesions under pneumothorax, and the cavities of the lower lobe not cured by the common collapsotherapeutic methods, are very well established indications in which the results are generally satisfactory. The other groups of indications were considered as our experience increased. The majority of these cases involve lesions where only resection of that diseased part of the lung could bring about a cure.

Results and postoperative complications of pulmonary resection generally vary according to the type of lesion, and, therefore, to the indication under which the operation was performed.

In Table III are set forth the relations between the operative indications, the postoperative complications, and the final results.

Old opened cavities under pneumothorax are a good indication for resection. Among 14 patients operated upon for this indication, 5 pneumonectomies and 9 lobectomies were performed, and only 2 patients presented postoperative complications, 1 being dissemination and 1 bronchial fistula and empyema. The 12 patients without postoperative complications are clinically cured. Of the other 2, one died and the other still has lesions with positive sputum.

The exudative lesions, as we expected, represent the poorest indication for resection. Also these lesions show poor results no matter what therapeutic method is used. The patients operated on presented severe lesions of the caseous type, which commonly end in death under any of the usual treatments. Of the 11 resections, 9 were pneumonectomies and 2 lobectomies, with the following results:

Four presented disseminations; 5 had reactivation of old foci; 3 developed empyema, and only 2 showed an uneventful postoperative course. Five patients died; 3 are alive but still have lesions and positive sputum; only 3 patients out of 11 are apparently well and have negative sputum.

Two pneumonectomies and 2 lobectomies were performed in a group of 4 patients presenting residual cavities under extrapleural pneumothorax. In the postoperative period there was 1 dissemination; 1 reactivation; 2 bronchial fistulas with empyema; and only 1 patient had no complications. Two patients are dead; 1 is alive but still has lesions and positive sputum; only 1 patient is well and negative from the bacteriologic viewpoint. The patients belonging to this group were operated on in the beginning of this series, when our experience was still small. Although resection under extrapleural pneumothorax might

TABLE III. RESULTS IN RELATION TO THE INDICATION FOR OPERATION IN PULMONARY RESECTION IN TUBERCULOSIS

INDICATIONS	NUMBER	TYPE OF RESECTION		POSTOPERATIVE COMPLICATION					FINAL RESULTS			
									DEATH			
		PNEUMONECTOMY	LOBECTOMY AND SEGMENTAL RESECTION	SPREAD	FLARE-UP	BRONCHIAL FISTULA AND EMPYEMA	EMPYEMA WITHOUT BRONCHIAL FISTULA	NO COMPLICATION	IN DIRECT RELATION TO THE RESECTION	OTHER CAUSES*	APPARENTLY WELL, SPUTUM NEGATIVE	ALIVE WITH DISEASE
Cavity under pneumothorax	14	5	9	1	–	1	–	12	1	–	12	1
Exudative lesions under pneumothorax	11	9	2	4	5	–	3	2	3	2	3	3
Cavity under extrapleural pneumothorax	4	2	2	1	1	2	–	1	2	–	1	1
Postthoracoplasty disease	12	5	7	–	–	3	–	9	1	1	10	–
Large cavity of the upper lobe	16	6	10	4	3	4	1	3	2	4	9	1
Lower lobe cavity	10	–	10	2	2	4	1	1	2	2	6	–
Extensive unilateral tuberculosis	24	24	–	1	5	3	3	13	1	2	18	3
Fibrosis, bronchiectasis, & bronchial tuberculosis	4	1	3	–	1	–	–	3	–	–	4	–
Upper lobe disease suitable for thoracoplasty	3	–	3	1	1	1	–	1	1	1	1	–
Tuberculoma or filled cavity	4	1	3	–	–	2	–	2	–	–	3	1
Tuberculosis of a single pulmonary segment	1	–	1	–	–	–	–	1	–	–	1	–

*See Mortality.

sometimes be more difficult technically, our impression is that it should be considered a good indication, offering at present better results.

Also the cavities under thoracoplasty are to be considered a good indication. In a group of 12 patients, 5 pneumonectomies and 7 lobectomies were performed. Bronchial fistulas with empyema occurred in 3 patients; the other 9 presented no complications. From those 12 patients, 2 died and 10 are clinically well. As happened in the other groups studied, some of the deaths are directly traceable to the resections; however, others are not. This fact will be studied later on.

Six pneumonectomies and 10 lobectomies were done in a group of 16 patients with large insufflated cavities of the upper lobe. In some cases, there were also lesions in other lobes, which was the reason for our pneumonectomy. These patients presented the following postoperative complications: 4 disseminations; 3 reactivations; 4 bronchial fistulas and empyema; and 1 empyema without bronchial fistula. Only 3 patients presented no complications. Six patients are dead; 1 is alive with lesions; and the other 9 are clinically cured.

Ten lobectomies were performed on 10 patients with lesions of the lower lobe; there were 2 disseminations; 2 reactivations; 4 bronchial fistulas and empyema; 1 empyema without bronchial fistula; and 1 patient presented no complications. Four patients died and 6 are clinically cured.

Twenty-four patients with extensive lesions, in one lung only, were treated by pneumonectomy, with the following results: 1 dissemination; 5 reactivations; 3 bronchial fistulas and empyema; and 3 cases of empyema without bronchial fistula; the evolution in 13 patients was without postoperative complications. Of these 24 patients, 3 died, 3 are living with lesions, and 18 are clinically cured.

One pneumonectomy and 3 lobectomies were performed on patients with bronchiectasis and bronchial tuberculosis. One case presented reactivation and 3 no complications. All 4 patients are cured.

On 3 patients with lesions of the upper lobe where we could start with thoracoplasty, 3 lobectomies were performed; we had 1 dissemination; 1 reactivation; 1 bronchial fistula and empyema, and 1 patient with no complications. Two patients died and one is cured.

Among 4 patients with "filled" and blocked cavities, we performed 1 pneumonectomy and 3 lobectomies. There were 2 bronchial fistulas and empyema, and 2 patients had no complications. One patient still has lesions and positive sputum, and 3 are clinically cured.

One segmental resection was carried out on a patient with lesions of a single pulmonary segment. There were no postoperative complications and the patient is cured.

COMPLICATIONS

Table IV shows the incidence of the main postoperative complications observed in our patients, and the evolution of the complicated cases.

TABLE IV. POSTOPERATIVE COMPLICATIONS* FROM PULMONARY RESECTION IN TUBERCULOSIS

TYPE OF RESECTION	NUMBER	SPREAD	FLARE-UP REACTIVATION	BRONCHIAL FISTULA AND EMPYEMA	EMPYEMA WITHOUT BRONCHIAL FISTULA	MISCELLANEOUS†	NO COMPLICATIONS
Pneumonectomy	53	6 (11.3%)	11 (20.7%)	7 (13.2%)	7 (13.2%)	3 (5.6%)	26 (49%)
Lobectomy and segmental resection	50	8 (16%)	7 (14%)	13 (26%)	1 (2%)	2 (4%)	22 (44%)
Total	103	14 (13.5%)	18 (17.4%)	20 (19.4%)	8 (7.7%)	5 (4.8%)	48 (46.6%)
Final Results in Relation to Postoperative Complication							
Apparently well, sputum negative		3 (21.4%)	6 (33.3%)	8 (40%)	4 (50%)	—	48 (100%)
Death		9 (64.2%)	6 (33.3%)	7 (35%)	3 (33.3%)	5 (100%)	—
Alive with disease, sputum positive		2 (14.2%)	6 (33.3%)	5 (25%)	1 (12.5%)	—	—

*Some patients presented more than one complication.
†Transfusion accident, pulmonary embolus, intestinal hemorrhage, cardiac failure.

TABLE V. PULMONARY RESECTION IN TUBERCULOSIS

	TOTAL (103 RESECTIONS)				UNTIL DECEMBER 1948 (27 CASES)				JAN. 1949 TO JAN. 1951 (76 CASES)			
	INCIDENCE		MORTALITY		INCIDENCE		MORTALITY		INCIDENCE		MORTALITY	
COMPLICATIONS	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	CENT PER
Dissemination	14	13.5	9	8.7	8	29.2	5	18.5	6	7.8	4	5.2
Reactivation of old foci	18	17.4	6	5.8	4	14.8	4	14.8	14	18.4	2	2.6
Bronchial fistula and empyema	20	19.4	7	6.8	10	37.0	4	14.8	10	13.1	3	3.9
Empyema only	8	7.7	3	2.9	3	11.1	2	7.4	5	6.5	1	1.3

The most severe complication was the dissemination of the tuberculous process. Its incidence was 13.5 per cent (14 cases) in the entire series, corresponding to 11.3 per cent (6 cases) for the pneumonectomies and 16 per cent (8 cases) for the lobectomies. Of the 14 patients who presented dissemination, only 3 (21.4 per cent) are at this time clinically cured with negative sputum; 9 patients (64.2 per cent) died and 2 (14.2 per cent) still present lesions and positive sputum.

Reactivation of old established foci was observed in 18 cases (17.4 per cent). After the resection, these patients had negative sputum for some time, and then showed exacerbation of foci formerly inactive. This reactivation may or may not be related to the resection. In some cases where resection was not followed by a corrective thoracoplasty, there was an exaggerated expansion of the remaining part of the lung, which may be the cause of the reactivation. However, in other cases, reactivation of old foci took place without apparent cause, as frequently occurs with tuberculous patients. The reactivation of old foci was more frequent in the pneumonectomy group (11 cases, 20.7 per cent) than in the lobectomy group (7 cases, 14 per cent). Of the 18 cases, 6 are cured (33.3 per cent), 6 are dead (33.3 per cent), and 6 still present lesions and positive sputum (33.3 per cent).

Empyema and bronchial fistula was the most frequent complication. Twenty cases were observed (19.41 per cent) consisting of 7 (13.2 per cent) pneumonectomies and 13 (26 per cent) lobectomies. Of these patients, 8 are cured (40 per cent); 7 died (35 per cent), and 5 (25 per cent) still have lesions and positive sputum.

Empyema without bronchial fistula was observed in 8 cases (7.7 per cent), 7 after pneumonectomies (13.2 per cent), and 1 (2 per cent) after lobectomy. Of these patients, 4 are cured (50 per cent); 3 died (33.3 per cent), and 1 (12.5 per cent) still has lesions and is bacteriologically positive.

Other complications indirectly related to the resection were observed in 5 cases (4.8 per cent). These were transfusion accidents, pulmonary embolus, intestinal hemorrhage, cardiac insufficiency, and 1 case of sudden death. These patients all died.

Of the entire series, only 48 patients (46.6 per cent) had no complications, 26 after pneumonectomies (49 per cent) and 22 after lobectomies (44 per cent). All of these patients are cured.

Postoperative complications presented a greater incidence in the early cases of our series, and at the moment they are less frequent. In the first 27 patients operated on, from February, 1946 to December, 1948, streptomycin was not used. It has been proved by various authors that this antibiotic decreases the number of complications and the mortality when administered during the pre- and postoperative periods.

Following 1948, based on our previous experience, we followed a more careful surgical technique; we systematically used the prone position, and the patients were prepared by the administration of 1.0 gr. streptomycin daily for fifteen days. As can be observed in Table V, complications from then on were less frequent and mortality correspondingly lower. Dissemination, which in

the early period represented an incidence of 29.2 per cent, with a mortality of 18.5 per cent, fell off to 7.8 per cent and 5.2 per cent, respectively. The same is true of the bronchial fistulas, in which cases the incidence decreased from 37 to 13.1 per cent and the mortality from 14.8 to 3.9 per cent. Empyema without bronchial fistula was also less frequent; its incidence was reduced from 11.1 to 6.5 per cent and the mortality from 7.4 to 1.3 per cent. Regarding the reactivation of old foci, it was noted that the incidence increased from 14.8 to 18.4 per cent, but the corresponding mortality decreased from 14.8 to 2.6 per cent. In 76 patients operated upon since January, 1949, there have been 40 (52.9 per cent) without postoperative complications, while in the previous series only 8 patients out of 27 (29.1 per cent) presented a postoperative evolution without complications.

TABLE VI. MORTALITY IN PNEUMONECTOMY FOR TUBERCULOSIS

Number of resections		53
Postoperative fatalities (1 month)	4	
Late fatalities (1 to 25 months)	8	
Causes of death:		
1. Spread	3	
2. Flare-up of old lesions*	2	
3. Empyema and toxemia	2	
4. Bronchial fistula, blockage of airway and asphyxia	1	
5. Transfusion reaction	2	
6. Pulmonary embolus	1	
7. Cardiac failure	1	
Deaths directly connected to the resection	6	(11.3%)
Deaths indirectly connected to the resection	4	(7.5%)
Deaths by new progressive lesions in apparently well patients	2	(3.7%)

*Apparently well with negative sputum 2 and 12 months after surgery.

MORTALITY

In Tables VI and VII are summarized all deaths observed during the entire follow-up period for these patients, that is, from nine months to five and one-half years. Many deaths are not directly related to the surgical process. Some were caused by accidents or intercurrent diseases; others were due to the evolution of new foci of tuberculosis in patients clinically cured of the lesions for which the pulmonary resections were performed. Among the 53 pneumonectomies performed, there were 6 (11.3 per cent) deaths directly related

TABLE VII. MORTALITY IN LOBECTOMY AND SEGMENTAL RESECTION FOR TUBERCULOSIS

Number of resections		50
Postoperative fatalities (1 month)	3	
Late fatalities (1 to 30 months)	10	
Causes of death:		
1. Spread	6	
2. Flare-up of old lesions*	4	
3. Empyema and toxemia	1	
4. Intestinal hemorrhage	1	
5. Suddenly (cause undetermined)	1	
Deaths directly connected to the resection	7	(14%)
Deaths indirectly connected to the resection	2	(4%)
Deaths by new progressive lesions in apparently well patients	4	(8%)

*Apparently well with negative sputum 2, 4, and 30 months after surgery.

to the operation and caused by postoperative complications—3 disseminations, 2 empyemas, and 1 opening of the bronchus in the immediate postoperative period followed by aspiration of the hemothorax. Four other deaths were indirectly related to the pneumonectomy (7.5 per cent), 2 of these deaths being due to transfusion accidents, 1 to pulmonary embolism, and 1 to cardiac insufficiency. Two patients (3.7 per cent) succumbed on account of the evolution of new pulmonary lesions, after they had shown negative sputum for two and twelve months, respectively.

The causes of death were also divided into three groups concerning lobectomies (Table VIII). In the first group, 7 deaths (14 per cent) were directly related to the resection—6 disseminations and 1 empyema and toxemia. In 2 cases (4 per cent) death was indirectly related to the surgical procedure and in 1 instance followed hemorrhage in intestinal lesions; the other patient died suddenly from an unknown cause. In the third group, four patients (8 per cent) died as a result of new lesions, after periods of two, four, and thirty months with negative sputum tests.

TABLE VIII. RESULTS (FOLLOW-UP, NINE MONTHS TO FIVE AND ONE-HALF YEARS) IN PULMONARY RESECTION IN TUBERCULOSIS

	PNEUMONECTOMY (53)	LOBECTOMY AND SEGMENTAL RESECTION (50)	TOTAL (103)
Clinically well and negative	34 (64.1%)	34 (68%)	68 (66%)
Radiologic lesions and positive	7 (13.2%)	3 (6%)	10 (9.7%)
Deaths directly connected to the resection	6 (11.3%)	7 (14%)	13 (12.6%)
Deaths due to other causes	6 (11.3%)	6 (12%)	12 (11.6%)

RESULTS

Table VIII summarizes total results observed in 103 cases. Sixty-eight patients (66 per cent) are clinically cured, with negative sputum, and are being observed for a period of from nine months to five and one-half years. Among these cured cases, there are 34 pneumonectomies and 37 lobectomies, corresponding to a percentage of cure for these two operations of 64.1 per cent and 68 per cent, respectively.

From a total of 103 patients, 10 (9.7 per cent) still present radiologic lesions and positive bacilloscopy. Among these patients, 7 had pneumonectomies (13.2 per cent) and 3 lobectomies (6 per cent).

Thirteen patients died (12.6 per cent) as a direct result of the resection, 6 of them being pneumonectomies and 7 lobectomies. The mortality was 11.3 per cent in the case of the pneumonectomies and 14 per cent for the lobectomies. Twelve other patients (11.6 per cent) died during this period of observation, due to causes indirectly related to the resection, with a percentage of 11.3 per cent and 12 per cent, respectively, for the 6 pneumonectomies and 6 lobectomies.

DISCUSSION

The statistical data gathered in this series enable us to analyze the problem of pulmonary resection in tuberculous patients. It must be kept in mind that

the first patients were operated on before the systematic use of streptomycin and therefore presented a great number of complications and deaths. The situation is now improved through the evolution of a better technique and the experience acquired in the matter, and a large number of the patients operated on have an evolution without complications.

Results are directly related to the operative indications. The best results were observed in open cavities under pneumothorax; in residual lesions after thoracoplasty; in cavities of the lower lobe; in fibrotic lesions without cavities and with specific bronchiectasis; and in "filled" and blocked cavities due to the closure of the drainage bronchus. In these cases, pulmonary resection offered a satisfactory number of patients clinically cured, accompanied by negative bacilloscopy. Also it was observed that the number of postoperative complications was less in these groups of patients.

In patients with extensive lesions localized in one lung, results were also very gratifying. All of these cases involved extensive lesions of long standing, well established and with little exudation. One technical detail that probably favored results in this group was the possibility of closing the bronchus of the diseased lung by means of special tubes and balloons, making it possible to perform the pneumonectomy without the danger of dissemination of tuberculosis to the other lung. This is not always possible when performing a lobectomy.

The poorest results in our series were observed among the patients with exudative lesions and caseous pneumonias under pneumothorax. These patients, with less established lesions, presented a great number of postoperative complications and a high mortality rate.

When the tuberculous process is limited to a single pulmonary segment, segmental resection generally offers good results. In this series of patients, operated on up to January, 1951, we had only one case of segmental resection. Since January, 1951, there have been other cases, which will be discussed in a later communication.

From a technical standpoint, many details could be discussed, as pulmonary resection in tuberculosis requires special care not necessitated in resections performed for other pulmonary diseases. Whenever possible, the bronchus corresponding to the part of the lung to be removed was blocked. This blockage was done by means of tubes carrying balloons, or double-lumen tubes. The Overholt prone position was always used, and we always took care to ligate the bronchus first. Whenever possible, before, during, or after the resection, a corrective thoracoplasty was performed. In some cases, the large distention of the remaining part of the lung was prevented by the use of pneumothorax. We do consider it important to protect the remaining lung. Of our patients, 17.4 per cent presented reactivation of old lesions, after a variable period of negative bacilloscopy, after the resection. In these patients, no protective thoracoplasty was performed to offset the hyperdistention of the lung. This is especially dangerous after pneumonectomies, due to the shifting of the mediastinum. In 20.7 per cent of our pneumonectomy patients on whom no posterior thoracoplasty was carried out, there developed reactivation of old foci within the remainder of the lung, after the absorption of the hemothorax.

TABLE IX. RESULTS (FOLLOW-UP FROM NINE MONTHS TO FIVE AND ONE-HALF YEARS) IN PULMONARY RESECTION IN TUBERCULOSIS

	TOTAL (103 CASES)	UNTIL DECEMBER 1948 (27 CASES)	JAN. 1949 TO JAN. 1951 (76 CASES)
Clinically cured	68 (66.0%)	13 (48.1%)	55 (72.3%)
Radiological lesions and sputum positive	10 (9.7%)	2 (7.4%)	8 (10.5%)
Mortality directly connected to the resection	13 (12.6%)	6 (22.2%)	7 (9.2%)
Mortality due to other causes, including new foci of tuberculosis	12 (11.6%)	4 (14.8%)	8 (10.5%)

Late results of pulmonary resections in tuberculosis remain precarious. A mortality of 12.6 per cent and a high morbidity of 53.4 per cent due to late postoperative complications would indicate this method to be inferior to other procedures of surgical collapsotherapy. Only 66 per cent of the patients of this series are clinically cured, whereas we know that the percentage of cures by other procedures can be higher. We must recall, however, that the results of the resections show constant improvement with the experience acquired, the latest patients in our series presenting fewer complications and a greater number of cures. From this point of view, we can divide our 103 cases into two groups (Table IX).

The first group includes 27 patients operated on up to and including December, 1948; these patients did not receive streptomycin routinely in the pre- and postoperative periods, and the surgical procedure in these cases did not include certain technical details which have now been introduced. Of these patients, 48.1 per cent are clinically cured; 7.4 per cent still present radiological lesions and positive sputum; 22.2 per cent died from causes directly related to the resection; and 14.8 per cent died from other causes, including those deaths due to new foci of tuberculosis.

In the second group are 76 patients operated on from January, 1949, to January, 1951. In this group, the patients all received streptomycin routinely, and there were 72.3 per cent of the patients clinically cured; 10.5 per cent with radiological lesions and positive sputum; a mortality of 9.2 per cent due to causes directly related to the resection; and 10.5 per cent of deaths due to other causes.

SUMMARY

1. One hundred three cases of pulmonary resections for the treatment of pulmonary tuberculosis were studied. These patients were operated on from February, 1946, through January, 1951, there being 53 pneumonectomies, 49 lobectomies, and 1 segmental resection. In a group of 76 patients operated on after December, 1948, streptomycin was administered routinely pre- and postoperatively. These patients showed better results than did those operated on earlier. The following data refer to these 76 patients.

2. The principal postoperative complications were:

(a) *Dissemination* in 7.8 per cent of the cases, more commonly observed after lobectomies than after pneumonectomies;

(b) *Reactivation* of old established foci in 18.4 per cent of the cases, more frequently found in pneumonectomies than in lobectomies;

(c) *Bronchial fistula and empyema* in 12.1 per cent of the cases, more often following lobectomies;

(d) *Empyema without bronchial fistula* in 6.5 per cent of the cases, more commonly after pneumonectomies.

3. Of the patients, 9.2 per cent died due to causes directly connected with the resection; mortality was less in pneumonectomies than in lobectomies. Of the patients, 10.5 per cent died due to causes indirectly related to the resection; included in this group are deaths due to new foci of tuberculosis after a period of negative bacilloscopy.

4. Of the patients, 72.3 per cent are clinically cured and have negative sputum, and 10.5 per cent still present radiological lesions with positive sputum.

5. The patients who showed the smallest number of postoperative complications and the largest number of cures were operated upon under the following indications: open cavities under pneumothorax, residual lesions after thoracoplasties, cavities of the lower lobes, and fibrotic lesions without cavities and with bronchiectasis and "filled" and blocked cavities.

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A SIMPLE TEST OF PULMONARY FUNCTION EMPLOYING THE OXIMETER

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IN RESPONSE to a constantly increasing demand for means of evaluating pulmonary function in patients with pulmonary disease, a great many testing procedures have been developed. The ventilatory mechanics of the lungs may now be adequately evaluated by measurements of vital capacity, maximum breathing capacity, breathing reserve, and a study of the breathing pattern.[1] Evaluations of the efficiency of gas exchange in the lungs are not as readily made.[2] Intensive analyses of pulmonary function of the type described by Riley and Cournand[3] are of great value in selected cases, but such procedures are well beyond the scope of routine tests to be employed in the average institution.

The oximeter has come to be recognized as a simpler means of obtaining information as to blood gas exchange, and many applications of this instrument have been described. Because of the obvious importance of the speed with which gases can be equilibrated between the inspired air and the blood, several have studied the time required for the change in hemoglobin saturation between air and oxygen breathing.[4-6] Unfortunately, tests of this type concern themselves with the flat portion of the hemoglobin dissociation curve where significant changes in blood oxygen tension correspond with changes of only a few percentage points in saturation, a circumstance demanding extreme accuracy of the measuring instrument. Inherent limitations in the method of ear oximetry[7] render such accuracy difficult, if not impossible, to attain. It would be much more desirable to work at levels of hypoxia where the steeper slope of the dissociation curve would minimize the significance of small instrumental errors. In addition, direct analytical methods have clearly documented the value to be derived from studying patients under conditions of mild hypoxia as well as under normal conditions of oxygenation.[8] We have therefore developed a method which offers the advantages of simplicity combined with a relatively high degree of accuracy to measure the rapidity with which a patient can resaturate the blood hemoglobin from the hypoxic condition. Aside from the oximeter, it requires no equipment other than that which is available in any hospital.

Apparatus.—This test requires a single-scale compensating oximeter of the Millikan type.* To avoid the inaccuracies of saturation changes over a very narrow range, a 10 per cent change in saturation was selected as the reference

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*We have employed oximeters manufactured by Coleman Instruments, Inc., Maywood, Ill., and by Waters Conley Co., Rochester, Minn. In general, we have found the Coleman instrument more satisfactory for routine testing purposes, although the Waters Conley instrument is required if deeply pigmented Negroes are to be tested.

index. Some degree of instability in the saturation readings may result from minor fluctuations in the exciter lamp voltage. We have therefore introduced a voltmeter with an easily read 3 inch scale into this circuit, together with a series resistance for adjusting the voltage. By setting this voltage slightly below the maximum obtainable, drifts in the battery voltage as indicated on the meter can be compensated by adjusting the resistance and thereby hold the intensity of the exciter lamp constant. This arrangement has the further advantage that with the occasional patient whose skin is too delicate to tolerate the normal heat of the lamp, a slightly lower voltage may be selected and thereby eliminate discomfort for the patient.

In addition to the oximeter, the test requires use of a spirometer equipped with a CO_2 absorber of the type used for basal metabolism recording, and some form of timing device. The respirometer as described by Cournand[1] is particularly well suited to this procedure, but any basal metabolism recording machine having a capacity of 6 liters or more may be used. It is desirable, though not essential, to have the cutoff valve of the spirometer built into the mouthpiece connector at the patient end of the breathing tubes, so as to be able quickly to switch the patient from spirometer breathing to room air breathing. Instruments with the cutoff valve located in the base of the spirometer would require removing the mouthpiece to accomplish this switch unless a correction factor is introduced for the dead space of the breathing tubes. Similarly, to facilitate changing the gas mixture in the spirometer without removing the mouthpiece from the patient, a pair of large three-way valves have been introduced into the breathing tubes through which the spirometer may easily be flushed and filled with room air. For timing purposes, any clock or watch with a sweep second hand may be used, but a stop watch or second indicator of the metronome type is preferable, since it permits the operator to keep his eyes fixed on the oximeter while the resaturation time is measured.

Procedure.—The patient is instructed to lie down and relax upon a suitable cot. The oximeter is turned on and adjusted, and the earpiece applied to the patient. A ten-minute interval must then elapse to permit warming of the ear and maximal dilation of the blood vessels.[9] We take advantage of this interval by using it to obtain vital statistics and a brief history from the patient. The patient is reassured as to the simple nature of the test and impressed with the fact that his only duty will be to relax and breathe naturally into the spirometer until the test is completed.

At the end of the warm-up period, the spirometer is filled with pure oxygen, the mouthpiece and noseclip applied, and the patient begins to breathe into the spirometer. For some edentulous patients, or others who find this method quite uncomfortable, a face mask may be substituted, providing the connections to the spirometer do not significantly increase the dead space. The voltage reading in the exciter lamp circuit is checked at intervals throughout the test, and kept accurately at the predetermined value. We usually run the lamp at 5.7 volts instead of the 6 volt maximum to permit adequate adjustment for battery drifts.

Unless severe functional deficiencies are present, the oximeter will usually be well stabilized at the end of three minutes of oxygen breathing at a level which, by adjustment if necessary, should read 100 per cent saturation on the oximeter scale. If at this time an upward drift of the oximeter persists, oxygen breathing is continued until a 100 per cent reading is maintained. The valve is then turned to cut the patient off from the spirometer and allow him to breathe room air. Within three to four minutes most patients will show a drop to a stable saturation level which is read on the oximeter and recorded as the *basal saturation*. With severe deficiency a longer time lapse may be required to achieve stabilization.

While waiting for stabilization, the spirometer is emptied and then filled and emptied three times with room air. With three-way valves in the breathing tubes this may be accomplished without disturbing the patient, otherwise it will be necessary to remove the mouthpiece in order to flush the spirometer. The spirometer bell is then filled with about 6 liters of *room air*. The exact volume required will vary with the volume of the dead space of the particular apparatus employed, but the total volume should be sufficient to allow the patient to consume about 1.5 liters of oxygen without danger of exhausting the gas volume in the spirometer bell on inspiration. An unnecessarily large initial volume of air in the spirometer serves only to prolong the test. After the spirometer has been flushed and properly filled with air and the patient stabilized at his basal saturation level, he is turned back for spirometer breathing.

It will be apparent that as the patient rebreathes from this air-filled spirometer, his oxygen consumption will progressively lower the oxygen tension in the system. For the first three to four minutes the hemoglobin saturation will fall very slowly. Then as the oximeter reading drops below 90 per cent, a progressively more rapid fall is observed. This fall is frequently cut short for a brief period by mild reflex hyperpnea. In any case, the oximeter is watched closely until the reading drops to 80 per cent saturation. The patient is then immediately cut off from the spirometer and switched to breathing room air. If the apparatus does not have a valve at the mouthpiece, the mouthpiece must be removed at this point to achieve immediate conversion to room air breathing. *The time is accurately measured for the return of the saturation from 80 to 90 per cent.* This time is recorded as the *resaturation time.*

This reading terminates the test unless a deficient resaturation time of more than twenty seconds is recorded. In that case we have found it advisable to recheck the setting of the oximeter to make certain that there is no instrumental error. This is accomplished by refilling the spirometer with pure oxygen and turning the patient back for spirometer breathing for another two to three minutes. Proper instrument standardization will be indicated by a return of the oximeter reading to 100 per cent. We regard a recheck value of over 98.5 per cent with oxygen breathing as evidence of a valid test; a recheck which stabilizes below this level with oxygen breathing indicates significant instrumental or technical error and the test should be repeated. Instrumental drifts of the order of 1 per cent between the beginning and end of the test, however, do not significantly alter the validity of the resaturation time determination.

In carrying out this test, it is obviously essential to maintain efficient absorption of CO_2 in the spirometer circuit at all times in order to exclude any complications due to hypercapnea. This necessitates changing the soda-lime absorber after every ten to fifteen tests, depending upon the capacity of the absorber.

Normal Values.—Many studies have been made of the basal hemoglobin saturation in normal adults. Although the results have shown some variation, most recent work has placed the normal saturation level at somewhere between 96.0 to 98.5 per cent. Thus Wood and associates[9] found in 35 normal adults an average value of 96.7 ± 0.3 per cent, while in 41 subjects Ferguson and coworkers[10] obtained an average of 96.0 ± 0.17 per cent. Greifenstein and associates[11] cited normal values in their laboratory of 97.4 ± 2.1 per cent. Drabkin and Schmidt[12] studied 5 normal subjects, and found all values to be above 98 per cent. Barach and Steiner[13] studied 8 normal subjects and 11 patients with

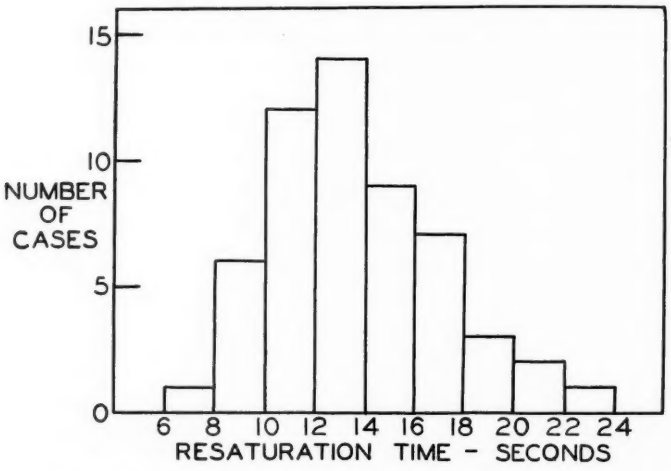

Fig. 1.—Resaturation time values (80 to 90 per cent) measured on 55 normal adults.

coronary artery disease, and in no case found a basal saturation below 96 per cent. In view of these findings, and on the basis of our own experience, we have set 96.0 per cent as the lower limit of what we regard as a definitely normal value for the basal saturation. Obviously the selection of a normal limit as high as this does not warrant the assumption that patients who fall slightly below this limit must necessarily have a pathologic deficiency. Nevertheless it indicates that a subnormal function level is being maintained which, when correlated with other findings, may appear to have clinical significance.

Normal values for the resaturation time recorded in this test were obtained from tests on 55 normal adults in good health who had no history of pulmonary or cardiac disease. This group was composed of 18 males and 37 females with an average age of 23 years, ranging from 17 to 46 years. No significant differences

were observed between the sexes or between the younger and older individuals in the resaturation times recorded. The results of this study are shown in Fig. 1. The mean resaturation time was 13.4 seconds with a standard deviation of ±3.4 seconds. On this basis a resaturation time of 20 seconds, which is approximately two standard deviations from the mean, has been selected as the upper limit of normal. Resaturation times between 21 and 30 seconds are considered suggestive of some pathologic deficiency and values above 30 seconds are considered to indicate definite pulmonary deficiency.

Results of the Test in Patients with Known Pulmonary Disease.—Table I summarizes the readings obtained upon 300 hospitalized patients with pulmonary tuberculosis. No specific selection was involved except for the exclusion of patients who showed clinical evidence of cyanosis. It should be emphasized that this test is designed specifically to detect unrecognized cases of deficiency in pulmonary gas exchange and is not applicable to patients with definite cyanosis in whom the basal saturation will be well below the 90 per cent level.

TABLE I. DISTRIBUTION OF TEST RESULTS ON 300 TUBERCULOUS PATIENTS

RESATURATION TIME (SECONDS)	BASAL-SATURATION (PER CENT)					
	100-98.0	97.9-96.0	95.9-94.0	93.9-92.0	91.9-90.0	TOTAL
Less than 21	17	123	28	1	0	169
21-30	1	29	17	1	0	48
31-60	2	22	13	6	0	43
61-120	0	6	12	6	0	24
More than 120	0	2	5	3	6	16
Total	20	182	75	17	6	300

In evaluating the results shown in Table I, consideration must be given to the pathologic physiology that may be involved in these cases. Riley and Cournand[3] have stressed the importance of maintaining a normal ventilation/perfusion ratio in the lung alveoli. Ventilation may be deficient due to a purely quantitative reduction in the volume of ventilation for either local alveoli or an entire lung. This would be observed in such conditions as therapeutic collapse, severe pleural thickening preventing adequate expansion of the lung, and to varying degrees in many types of infiltrative processes. Such a pure *ventilatory* defect would obviously result in inadequate aeration of the blood perfusing the alveolar capillaries and thus act to lower the basal saturation. Nevertheless, when confronted with a sudden change in the inspired oxygen tension, there is no barrier to rapid gas diffusion in such lungs and therefore the resaturation time would be influenced to only a minor degree.

In contrast, ventilation may be interfered with in a qualitative manner by imposing a barrier to the diffusion of gases between the inspired air and the lung alveoli. This defect is observed classically in emphysema with its large dead spaces interfering with rapid diffusion, and would also be observed to varying degrees in the presence of any fluid barrier to gas diffusion as occurs with edema and many exudative types of disease. These *diffusion* defects will tend to lower the alveolar oxygen tension somewhat. However, since hemoglobin

saturation does not drop significantly until there has been a marked reduction in the alveolar oxygen tension, the basal saturation will not be lowered significantly until the defect becomes severe. However, when presented with the problem of resaturation from the hypoxic condition, a patient with a barrier to efficient gas diffusion should exhibit a significant prolongation of his resaturation time.

To evaluate the possibility of differentiating these conditions with the oximeter test employed here, oximetry findings were compared with a careful analysis of breathing patterns for evidence of functional emphysema. As described by Cournand,[1] breathing patterns which show abnormal prolongations of deep expirations, inability to maintain a rapid rate of breathing, and a tendency for the expiratory position to ride up toward the inspiratory position with successive deep or rapid respirations were considered as mechanical evidence of significant functional emphysema which might be expected to be associated with diffusion defects. Of the 32 patients in Table I who showed saturation times greater than 30 seconds but basal saturations above 96.0 per cent, all 32 showed marked evidence of emphysema in their respiratory patterns. None of these patients were found to have had any therapeutic collapse. These findings are in accord with what would be anticipated in the presence of a diffusion defect.

On the other hand, patients with basal saturations below 96 per cent but with resaturation times of less than 21 seconds should represent those with simple ventilation deficiencies. Actually 25 of the 29 patients in this latter group were found to have had therapeutic collapse of one or both lungs when the test was made. Only 7 patients exhibited respiratory patterns which we would consider to be suggestive of some emphysematous disturbance, and in none of these was the condition marked.

In the remaining deficient groups, no consistent correlation was observed between clinical findings, respiratory patterns, and oximetry readings which would justify the use of the oximetry readings to differentiate the nature of the pulmonary disturbance. On this basis, of the total of 160 patients who were considered to have some deficiency in their oximetry, 99 or 62 per cent were found to have deficiencies of an apparently mixed type which could not be differentiated further, while in 38 per cent the deficiency could be identified as being either of the ventilatory or the diffusion types. In terms of the total tuberculous population of 300 patients tested, 47 per cent had no oximetric deficiency, 33 per cent had deficiencies of a mixed type, and 20 per cent had deficiencies which could be differentiated as either ventilatory or diffusion in nature.

Reproducibility.—The reliability of any test must be established in terms of its ability to give equivalent values when the same subject is retested. We have found it impractical to attempt to perform two complete tests in succession upon the same patient because of the progressive discomfort of the mouthpiece and noseclip and the drying of the mouth which results from spirometer breathing. A series of 15 patients was selected, therefore, whose disease was stable enough so that changes in pulmonary function would not be

expected to occur over short intervals. Retesting of these patients, at an interval of a few days to several weeks after the initial test, revealed very good agreement between the values of the two tests as shown in Table II. As illustrated by the last case in this series, and as we have observed not infrequently in subsequent experience with the test, patients with extremely prolonged resaturation times sometimes exhibit considerable quantitative variation in the actual resaturation time values, but in no case have such variations obscured the presence of a marked deficiency in each test.

Critique: Technical.—The outstanding feature of this test is its simplicity. A technician acquainted with basal metabolism testing can learn how to perform this test in a single day. The entire test, from the time the patient enters the

TABLE II. RESULTS OF RETESTS UPON THE SAME PATIENT

PATIENT	DATE	BASAL SATURATION (PER CENT)	RESATURATION TIME (SECONDS)
J. R.	7/28/49	96.8	11
	7/29/49	96.2	11
R. L.	10/25/49	96.7	12
	11/ 1/49	96.4	11
S. C.	2/10/50	96.9	11
	2/20/50	96.5	12
W. W.	3/28/50	97.2	15
	4/19/50	97.0	18
	4/26/50	96.8	15
T. H.	8/23/49	96.9	17
	10/12/49	97.0	16
R. F.	4/ 5/50	97.1	16
	4/18/50	97.1	20
C. B.	1/26/50	96.5	19
	2/14/50	96.5	21
P. F.	6/16/49	95.2	23
	8/23/49	96.1	22
R. S.	11/16/49	97.1	22
	1/25/50	96.4	27
R. J.	2/ 2/50	96.0	26
	3/ 7/50	96.5	23
G. M.	2/16/50	96.0	38
	2/27/50	96.2	27
R. C.	1/12/50	96.9	59
	3/15/50	96.8	50
D. K.	3/13/50	95.3	62
	3/23/50	94.9	55
H. G.	2/10/50	96.4	110
	2/12/50	96.9	101
L. W.	11/15/49	95.3	158
	1/30/50	94.8	69

laboratory until the final report is filled out, requires about 30 minutes. From the standpoint of the patient, it entails little more than the standard basal metabolic rate test which it closely resembles. Subjective symptoms of hypoxia are minimal. In contrast to many tests of pulmonary gas exchange which require breathing according to specific instructions, this test involves only natural respiration. It therefore is not dependent upon any significant degree of patient cooperation or patient intelligence. Small leaks around the mouthpiece or face mask, which constitute a major hazard in many pulmonary function tests, are of no importance provided that the leaks are not so great as to prevent attainment of the 80 per cent saturation level. This test may be carried out at the bedside of the patient who is acutely ill as well as on the ambulatory patient. Indeed we have encountered many instances where acute complications such as hemoptysis and severe chest pain have contraindicated the usual ventilatory tests, and oximetry appeared to be the only functional test that could be performed with safety.

A particular feature of safety in this test is that the degree of reduction in the oxygen tension in the inspired air is automatically determined by the patient's own functional capacities. Analyses of the oxygen content in the spirometer at the 80 per cent saturation level have revealed that, in the patient with severe functional impairment, this level may be reached at 13 to 15 per cent oxygen; while in others the inspired oxygen may fall to as low as 7 per cent before the 80 per cent saturation level is reached. One thereby avoids the possibility of precipitating an anginal attack in a patient with severe cardiopulmonary disease who is exposed to a predetermined level of restriction in the inspired oxygen tension.[14] It is for this latter reason that we terminate the hypoxia at the entirely safe level of 80 per cent saturation.

A source of variation in the resaturation times observed in normal subjects (Fig. 1) resides in the variable degree of respiratory stimulation associated with mild hypoxia in different subjects.[15, 16] Since the initial portion of the resaturation process is strongly influenced by the exact depth and rate of the first few breaths after the switch-over to air breathing, the pattern of the initial rise in the oximeter reading is quite variable. In addition, most individuals exhibit a posthypoxic depression in saturation, such that, after the return to room air breathing, the oximeter will rise to only 94 or 95 per cent and then often fall a point or two before slowly climbing to the original basal saturation level. The 80 to 90 per cent interval was therefore selected as the optimal index to resaturation, since it reduces the importance of the initial portion of the process but still stays comfortably below the posthypoxic depression in saturation. On this same basis, however, it is important to recheck the 100 per cent saturation setting on the oximeter when delayed resaturation times are observed. Errors of greater than 1.5 per cent in the oximeter reading could result in a confusion of the normal posthypoxic depression in saturation with an apparent prolongation of the 80 to 90 per cent resaturation time.

In spite of its variability, the respiratory stimulation during the hypoxic exposure is usually not more than 50 per cent above the basal ventilation rate. In occasional subjects, however, significantly greater degrees of hyperpnea are

observed which can introduce complications into the resaturation time determination. When such individuals are returned to air breathing, some will demonstrate a prompt acapneic depression of respiration which will tend to prolong the resaturation time. Others carry the hyperpnea over through the resaturation period and thereby abbreviate the resaturation time. In our experience, errors from this source do not become significant until hyperpnea in excess of 20 liters per minute is encountered. We therefore have adopted the practice of calculating the ventilatory rate at the end of the hypoxic period from the spirometer record. If the minute volume of respiration at this time is over 20 liters, the test is considered invalid and repeated. It should be understood that retesting for this reason is required in only a very small percentage of patients; nevertheless, in evaluating individual patients, a serious error might be made if complications of this nature were not recognized.

Critique: Theoretical.—When used for the routine evaluation of patients, it is obvious that this test in itself has limited value until it is correlated with the over-all clinical and laboratory findings. Like most tests of this nature, it does not indicate whether pulmonary deficiencies are due to primary pulmonary disease or to pulmonary disturbances secondary to cardiovascular disease. Its interpretation is also conditioned by the assumption that there are no abnormal vascular shunts by-passing the pulmonary bed and no abnormalities in the blood hemoglobin to invalidate the oximeter method.

The ability of this test to distinguish whether deficiencies are dominantly due to simple ventilatory defects or to diffusion defects cannot be compared in accuracy to the information that can be obtained by direct analysis of alveolar and blood gases.[3] On the other hand, it must be recognized that in many chronic lung diseases one would expect to find both types of deficiency present. Therefore the large proportion of patients with tuberculosis who showed mixed oximetric deficiencies (see Table I) is not unreasonable. From a clinical standpoint, it is the more extreme examples of the two types of deficiency which it is most important to identify, and it is felt that the test described here offers some real assistance in making this type of differentiation.

Originally it had been hoped that the interpretation could be carried somewhat further by obtaining more data upon respiration at the hypoxic level. Theoretically it should be possible to calibrate the system so that the amount of oxygen consumed by the patient, as indicated by the spirometer record, could be converted directly into the tension of oxygen in the inspired air at that time. This, correlated with the ventilation rate and saturation, should throw further light upon pulmonary gas exchange. Actual gas analyses together with pneumographic studies of changes in chest volume, however, revealed that changes in the tonus of inspiratory muscles associated with hypoxia[17, 18] so distort the volume of the lung-spirometer system as to invalidate such an analysis of the recordings.

The exact degree of significance which should be attributed to the mild deficiencies detected by this test is not always clear. In considering patients for pulmonary resection, for example, it is encouraging to find oximetry readings that are within normal limits. Such findings are not at all uncommon in

those cases where the diseased area has been essentially excluded from contributing to pulmonary function and where secondary processes have not resulted in diffuse damage to the remaining lung. On the other hand, in those cases where deficiencies in the oximetry readings are observed, there is as yet no adequate basis for assessing how severe a deficiency should be present in order to contraindicate major chest surgery. In our own experience, however, we have found this oximeter test, when weighed together with the over-all clinical picture, of considerable practical value, as may be best illustrated by the brief case reports given here.

ILLUSTRATIVE CASES

CASE 1.—R. K., a 43-year-old white man, had known pulmonary tuberculosis over a period of eight years with bilateral apical cavitation. Pneumoperitoneum, right phrenic paralysis, streptomycin, and PAS had served to control the disease considerably and closed the cavity on the right. The cavity in the left upper lobe persisted, and the patient was considered for major surgery.

Clinically this patient appeared to be only a fair operative risk. His appetite was poor and he was underweight. Chest roentgenogram revealed, in addition to the cavity on the left and dense scarring in the right apex, considerable scattered mottling throughout both lung fields with numerous stringy shadows.

In the laboratory he was found to have a vital capacity of 3.1 liters or 70 per cent of normal. Maximum breathing capacity was 61 liters per minute. The respiratory pattern showed definite abnormalities interpreted as loss of normal lung elasticity. Oximetry revealed a basal saturation of 96.8 per cent and a resaturation time of 90 seconds, indicating a severe deficiency of the diffusion type. On this basis the clinical impression of a fair operative risk was revised to that of a poor operative risk and major surgery was deferred.

This case illustrates a common problem in chronic pulmonary tuberculosis. Deficient pulmonary function must always be suspected in the presence of radiological evidence of diffuse fibrosis, but we have observed many such cases in which pulmonary function remained reasonably good. Knowledge of the functional significance of these fibrotic shadows, as obtained by the oximeter test, may aid considerably in formulating the proper course of treatment.

CASE 2.—R. H., a 21-year-old white man, had a right lower lobectomy two years previously for bronchiectasis. Pathologic examination of the specimen revealed the surprising findings of caseation necrosis and acid-fast bacilli. On this basis the right hemidiaphragm was paralyzed and the patient placed on pneumoperitoneum therapy and bed rest. After a few months on this regime, the patient left the hospital against medical advice and continued the pneumoperitoneum under the care of a private physician. One year later he was admitted to Brecksville Hospital with hemoptysis. Examination revealed bilateral spread of the disease, with dense pneumonic disease and cavitation throughout the right upper and middle lobes and scattered infiltrations throughout the left lung. A course of streptomycin therapy controlled the disease on the left and led to some resolution on the right, although dense infiltration with probable cavitation persisted. The patient was in fair general condition, but complained of some shortness of breath on mild exertion.

Laboratory examination revealed a vital capacity of 1.9 liters or 46 per cent of normal and a maximum breathing capacity of 69 liters per minute. Oximetry yielded a basal saturation of 95.6 per cent and a resaturation time of 16 seconds. The slightly low basal saturation was attributed to a ventilatory defect associated with the pneumoperitoneum. The normal resaturation time was taken as evidence that there was no significant physiologic damage in the functioning left lung.

It was elected to perform a thoracoplasty on the right. The postoperative course was uneventful. Three months postoperatively the ventilatory capacity showed a significant increase, associated with the greater expansion of the left lung due to discontinuance of the pneumoperitoneum. The basal saturation had risen to 96.9 per cent and the resaturation time was 11 seconds.

This case represented a difficult clinical decision. Some would favor radical resection of the remaining right lung. Others, in view of the past history including recent disease scattered throughout the left lung, would hesitate to embark upon major surgery. Knowledge of the functional status of the left lung, as obtained by the oximeter, can aid in making the decision.

CASE 3.—H. M., a 51-year-old white man, had a left pneumonectomy two years previously for carcinoma. Since then the patient had complained of severe dyspnea. His local physician had informed him that he suffered from compensatory emphysema of the remaining lung and that there was nothing that could be done for the condition. On this admission the patient was markedly dyspneic, orthopneic, and had become a respiratory cripple. Cyanosis was absent. Cardiac findings were negative and there was no evidence of a recurrence of malignancy. X-ray examination of the chest showed some mediastinal shift and distention of the right lung without definite emphysema.

Laboratory study revealed a vital capacity of 2.0 liters or 64 per cent of normal and a maximum breathing capacity of 70.6 liters per minute. Oximetry gave a basal saturation of 95.2 per cent and a resaturation time of 16 seconds. It might be noted that at the start of this test the patient's attention was called to the fact that "the machine was filled with pure oxygen" and no dyspnea was observed during the oximeter test.

It is clear that the remaining right lung in this patient had undergone some degree of distention, but the finding of a resaturation time that was well within normal limits is considered to exclude any significant degree of functional emphysema. The dyspnea must therefore be explained in terms of a respiratory neurosis. This possibility had been entertained clinically, but, with a known etiological factor for emphysema present, it would have been difficult to deny a pulmonary basis for the crippling respiratory distress without the evidence of the oximeter.

It might be noted that we have encountered a number of similar cases in which the oximeter test has been invaluable in establishing the presence or absence of a pulmonary basis for a complaint of dyspnea. Unlike ventilatory tests, these oximeter readings cannot be distorted by lack of effort, willful or otherwise, on the part of the patient.

SUMMARY

1. A test is described for evaluating the efficiency of pulmonary gas exchange, employing the earpiece oximeter and a basal metabolism machine. After obtaining the basal saturation of the patient while breathing room air, his saturation is lowered to 80 per cent by having him rebreathe into the air-filled spirometer. The patient is then quickly switched to room air breathing and the time is measured for the saturation to rise from 80 to 90 per cent.

2. Measurements on 55 healthy adults indicate a normal resaturation time as determined by this test of 13.4 ± 3.4 seconds.

3. Results of this test upon 300 patients with pulmonary tuberculosis but without clinical cyanosis are reported. Deficiencies were demonstrated by this

test in 53 per cent of these patients. In 38 per cent of the patients exhibiting deficiencies, it was possible to estimate whether the deficiency was dominantly due to reduced ventilation or to impaired gas diffusion.

4. Retesting the same patients revealed adequate reproducibility of the test values for the routine use of the test in patient evaluation.

5. Technical and theoretical limitations of the method are discussed. The clinical use of the method is illustrated with case histories.

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SUCCESSFUL CONSTRUCTION OF AN ARTIFICIAL ANTETHORACIC ESOPHAGUS

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IT IS THE purpose of this paper to report further on a method of replacement for an esophagus, originally reported by Longmire and Ravitch,[1] which will occasionally be found useful. Reconstruction was necessary because of a severe stricture of the esophagus complicated by perforation, mediastinal abscess, esophageal-mediastinal-bronchial fistula, and chemical burns of the upper portion of the stomach.

The discomfort, inconvenience, and complications attendant upon gastrostomy and/or repeated esophagoscopy and bouginage make patients with a severely stenotic esophagus desirous of reconstruction. Prevailing methods use the stomach, jejunum, or tubes of skin. The stomach or the jejunum may be brought up either inside or outside the thorax.

If the distribution of the blood supply to the proximal jejunum is satisfactory, the method of Roux,[2] popularized by Yudin,[4] will be found best for routine use. Placing the stomach in the chest is not physiologic and is not well tolerated in older patients where gastric dilatation may produce uncomfortable fullness or induce episodes of cardiac irregularity. When the arrangement of the blood supply of the jejunum precludes the Roux[2] procedure and a previous injury prevents the intrathoracic transfer of the stomach, as in the patient to be reported, the operation described by Longmire and Ravitch[1] will be found useful. Based on experiments in dogs by Stafford,[3] the technique consists of transplantation and migration of a segment of jejunum in the subcutaneous tissues to effect a new union between the esophagus and stomach. The method is well described and beautifully illustrated in the paper of Longmire and Ravitch.[1]

CASE REPORT

L. W. (R-26,269), a 25-year-old Negro man, was admitted to the medical service of the Veterans Administration Hospital, Fort Howard, Md., with a history of swallowing a corrosive liquid four days previously. A Levin tube was easily passed into the stomach for feeding, and removed eight days later when the patient swallowed with minimal discomfort. At this time, demonstration of an esophageal-bronchial fistula and a mediastinal abscess by roentgenogram after the administration of Lipiodol (Figs. 1 and 2) made gastrostomy necessary. The fundus of the stomach was found to be severely burned so the gastrostomy was done near the pylorus. Later, the mediastinal abscess was drained extrapleurally after resection of about 5 cm. segments of the right eighth, ninth, and tenth ribs. The fistula was found at the level of the azygos vein. The opening in the esophagus was sutured and the closure reenforced by fascia from the latissimus dorsi muscle. The wound, which was left open, healed uneventfully.

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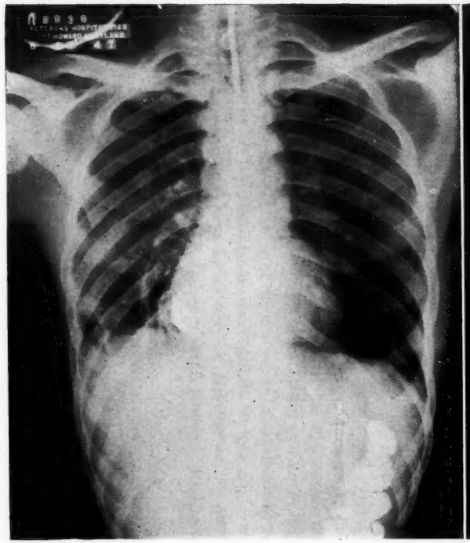

Fig. 1.

Fig. 2.

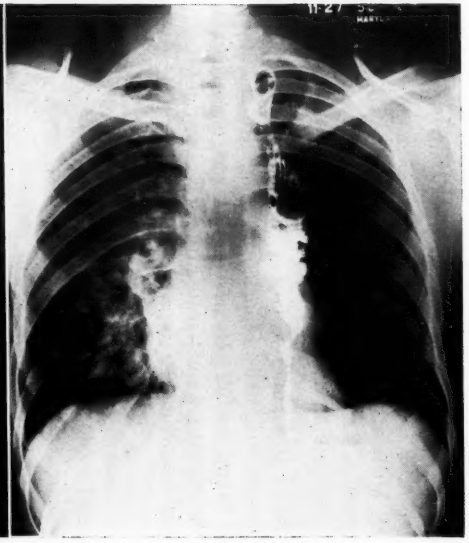

Fig. 3.

Fig. 1.—Demonstration of mediastinal abscess and bronchogram of lower lobe of right lung.
Fig. 2.—Demonstration of fistula between esophagus to mediastinum to lung.
Fig. 3.—Roentgenogram of antethoracic replacement after administration of barium.

Damage to the esophagus was severe, as demonstrated by the progressive stricture which ultimately extended to a point about 1½ cm. above the clavicle. Attempts to pass a piece of black silk through the esophagus were fruitless. Performance of a minor plastic procedure on the gastrostomy afforded an opportunity for retrograde bouginage but an obstruction was encountered at the esophagogastric junction.

Since some type of reconstruction was desirable, the conditions present made an antethoracic replacement using a jejunal graft the procedure of choice. At laparotomy, a segment of jejunum about 30 cm. in length, with two centrally located arteries and veins, was isolated, starting about 16 cm. below the ligament of Treitz and placed outside the abdomen. Intestinal continuity was restored by end-to-end anastomosis. A later complicating episode of acute intestinal obstruction was relieved by operative division of adhesions constricting the jejunum about 30 cm. below the anastomosis. A series of several operative procedures was necessary in order to cover the loop of jejunum with skin, divide the central pedicle, delay each end of the graft to insure an adequate blood supply, transfer the graft, close the

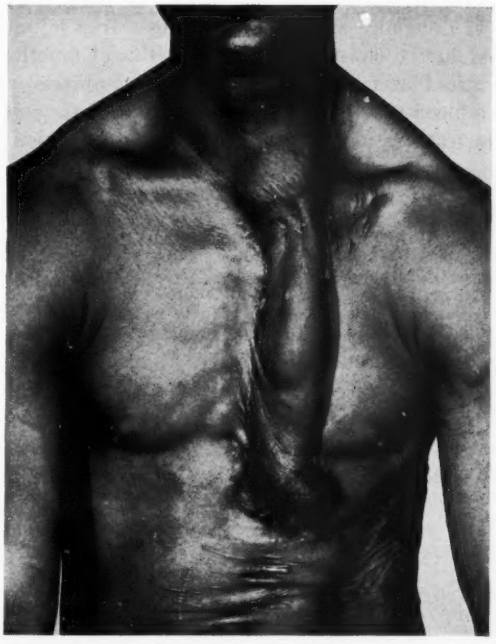

Fig. 4.—Patient about one and one-half years after discharge.

gastrostomy, and restore continuity of the alimentary tract. Despite creation of an apparently adequate anastomosis between esophagus and jejunum, a stricture developed and was corrected surgically. The patient was now able to eat a normal diet and was discharged from the hospital.

Approximately one month later, readmission for treatment of stricture again at the proximal anastomosis was necessary. A catheter for feeding purposes was inserted through an opening in the upper part of the loop of jejunum and a minor revision of the anastomosis was done subsequently.

Stricture formation, however, again returned necessitating further revision. The anastomosis and surrounding scar tissue were excised. Resection of the medial 3 cm. of the clavicle facilitated the making of a large end-to-end anastomosis. The postoperative course was uneventful. The patient has remained well for about one and one-half years. (Figs. 3 and 4.)

COMMENT

The procedure provides an isoperistaltic tube lined with mucous membrane as a replacement for the esophagus. A more physiologic passage was provided than by the Roux procedure since the stomach is not by-passed and the risk of formation of a gastric or duodenal ulcer is, therefore, seemingly reduced. A jejunal ulcer from regurgitation of acid gastric contents might result but the isoperistaltic movements of the jejunum should minimize this hazard.

The major objection to the procedure is the time required for performance which limits its use to benign diseases of the esophagus. Patience is necessary since stenosis of the anastomosis and fistulas are troublesome although solvable problems.

SUMMARY

The method of Longmire and Ravitch[1] using a free jejunal graft was successfully employed in the construction of an artificial antethoracic esophagus. Esophageal-mediastinal-bronchial fistula, mediastinal abscess, chemical burn of the stomach, and a blood supply to the proximal part of the jejunum precluding transposition were features making this type of reconstruction advisable.

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